# OPHTHALMOSCOPIC DIAGNOSIS

# OPHTHALMOSCOPIC DIAGNOSIS

BASED ON

TYPICAL PICTURES OF THE FUNDUS OF THE EYE
WITH SPECIAL REFERENCE TO THE NEEDS OF
GENERAL PRACTITIONERS AND STUDENTS

BY

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*WITH 86 COLORED PICTURES ON 48 PLATES AND 18 ILLUSTRATIONS
IN THE TEXT*

To

THE MEMORY OF

MY HONORED TEACHER,

JULIUS v. MICHEL

# Preface

THIS book is dedicated to the memory of *Julius v. Michel*, to whom is due the credit of its conception, its purpose, and its arrangement. I have simply followed out his idea in bringing into bold relief the relations that exist between diseases of the eye and those of the general organism. He had the satisfaction, which falls to the lot of few, to see the ideas, for which he had fought all his life, receive general recognition in his old age. His views concerning the material part played by tuberculosis in the etiology of diseases of the eye, as well as those in regard to the diagnostic importance of changes in the vessels of the fundus, are now generally accepted as correct. To a much less degree is this true of his theory concerning myopia, and the writer is well aware that he may excite dispute when he undertakes to present this conception in the present book.

In the title the word "Atlas" has been intentionally avoided, because it emphasizes the illustrations; the words "Ophthalmoscopic Diagnosis" have been chosen instead in order to indicate that the real purpose of the book is to be a systematic guide to diagnosis, and that the illustrations are intended to serve simply as aids in the carrying out of this purpose. The manner in which the text has been written and arranged has also been made subservient to this point of view. A glance at the Table of Contents will show that the ophthalmoscopic pictures of hemorrhages, white spots, black spots, etc., have been utilized solely as a means of classification, and that the attempt has been made to bring out the diagnosis, and to impress the clinical picture through the symptoms there depicted. The earlier text-books on ophthalmoscopy, such as those by *Jaeger, Mauthner, Schweigger, Dimmer, Schmidt-Rimpler*, and others, started from the clinical conceptions of disease, and portrayed these, with their details and symptoms. The first to take the ophthalmoscopic symptom as a basis for classification—at least in literature, for doubtless many besides ourselves had previously used it in teaching—was *Elschnig*, in his article on ophthalmoscopic differential diagnosis in *Axenfeld's* text-book. I have followed his lead in many places where his method of presentation seemed to be suitable for my purpose.

Special attention has been paid to those diseases of the eye that are related to general diseases, and the greatest consideration has been given to the varied needs of the general practitioner, the neurologist, the gynecolo-

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gist, and the syphilologist, in the manner in which they are presented. The reader will find in the Index not only the individual symptoms, but also connected with them the general diseases of which they form the ocular signs.

Pathology is entered into only so far as seemed advisable for the explanation of the ophthalmoscopic pictures. Brief space is likewise given to prognosis and treatment. Most of the pictures were taken with the aid of *Thorner's* demonstration ophthalmoscope, but they have been reduced about two thirds in size for reproduction until they present the inverted image magnified about ten times.

I cannot conclude without expressing my obligations to those who have helped me in this work. Above all I wish to show my gratitude to **Prof.** *Krueckmann*, who undertook the great labor of revising the manuscript, and to thank my colleagues who have been of great assistance by selecting and furnishing me with patients.

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cannot be taught in full detail in such a book as this, but a few suggestions may be of aid to those who are inexperienced.

## I.  INVERTED  IMAGE

### 1. Focussing upon the Papilla.

The light should be placed behind and to the side of the patient, so that it will not shine into his eye.

The complaint is heard very often in every class in ophthalmoscopy that the student can focus quite well upon the papilla of the left eye of the patient, but not upon that of the right.  The reason for this is that the patient obeys too literally the customary direction, that he should look at the corresponding ear of the observer.

The object of this direction is to cause the patient to look past the eye of the observer, or the mirror, at a certain distance, i.e., 15 cm, with his own eye slightly inclined toward his nose, in order that the papilla, which lies to the nasal side of the macula, may confront the observer.  But when the physician holds the mirror before his *right* eye, the distance between it and his *right* ear is considerably less than the requisite 15 cm: it is a **handbreadth,** some 10 cm too short, while the distance from the left ear to the mirror is about right.  It is therefore necessary that on the side on which the physician holds the mirror the patient should look **not at** the ear of the observer, but past it at the distance of about a handbreadth, in order to bring his papilla into view.

Furthermore, it is not advisable to have the patient look **at** the ear of the observer, because he then accommodates, and a contraction of the pupil accompanies accommodation: therefore it is better, even when the patient is sitting on the opposite side, to direct him to look not at the ear, but past it, as though at an object in the distance.  The direction usually given should therefore be made more precise, so as to read:

*In order to present his papilla the patient is to look at a distance past the ear of the observer corresponding to the eye that is being examined: if the latter is on the side on which the physician holds the mirror, he should look about a handbreadth away from the ear, if it is on the other side, the line of vision should pass close to the ear.*

The correct presentation of the papilla is a matter of the greatest importance, although it is generally undervalued by beginners. It is perhaps the most important point connected with the examination, for it may be said without exaggeration that in about three quarters of all the cases a failure on the part of anyone using the ophthalmoscope is to be ascribed to an incorrect, or inexact presentation of the papilla, or to an incorrect, or inexact direction to the patient where to look. Let it once be realized that the retina comprises an area of several square centimeters, that within that surface the papilla is only a minute point, not much larger than the head of a pin, and it will be appreciated that a considerable degree of accuracy is necessary to properly present this point in the relatively large surface.

*Hence it should be the rule in all cases in which the attempt to see the papilla is not immediately successful, to interrupt the examination and to ascertain first of all whether the position of the patient's eye is correct.*

After the patient has been told in what direction to look the observer at first closes the eye which is not at the ophthalmoscope and throws light into the eye of the patient without the interposition of a lens. By doing this he learns two things: 1, whether the refractive media are clear, or opacities are present in the cornea, lens, or vitreous, which may interfere with the examination; and, 2, whether the papilla is actually before him. If the patient presents his papilla correctly the pupil does not appear to be as red as it otherwise does, but is of a whitish yellow, because the tone of its color is determined by that of the papilla.

When this has been ascertained to be the case a lens is placed before the eye and the observer tries to obtain a sharply defined image of the papilla. To attain this end

## 2. The Correct Distance

between the optical systems, eye—lens—eye, is of paramount importance. When a +13 D lens [1] is used the distances are as follows:

The total distance between the physician and the patient is approximately 40 to 45 cm (see Fig. A in the text), of which 7 cm are between the patient and the lens, 32 to 37 cm between the lens and the physician. This is provided that both the physician and the patient are emmetropic. If either one

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[1] The strength of a convex lens may be ascertained in a very simple manner by producing the picture of any source of light upon a piece of paper by means of the lens. The distance at which the latter must be held from the paper in order to obtain a sharply defined image corresponds in general to the focal distance, which is measured in centimeters. Then it is only necessary to divide 100 by this number (100 cm = 1 m; the lenses are numbered according to the metric system) in order to learn its strength in diopters. If the image is sharply defined at the distance of 5 cm the lens is $\frac{100}{5} = 20$ diopters strength.

or both have over 4 D of myopia the physician must come closer; if either
one or both are quite hypermetropic the distance must be somewhat greater.
We must proceed in a manner similar to that employed in the use of a micro-
scope. First comes the gross presentation at the distances given above, and
then comes the micrometer screw, by the movement of the head of the physician
a little backward and forward until the image is sharply defined.

In high degrees of hypermetropia, like such as are present after removal
of the lens for cataract, the observer must increase the distance quite a good



Fig. A.

deal in order to obtain a distinct image of the fundus (see Detachment of
the Retina, page 152).

Next to faulty accommodation, incorrect distance is the most important
cause of indistinctness of the image (see under 6).

## 3. Correct Accommodation.

The image of the fundus produced by the convex lens lies in front of the
latter at the distance of its focal point, 7 cm in front of it, at point $B$ in
Fig. B. The physician must focus his eye upon this point. This is done
most easily when the point is at his ordinary reading distance, 25 to 30 cm,
and the accommodation used for reading is called into activity. But begin-
ners usually try to see the image in the eye of the patient, accommodate
incorrectly, and so cause the image to be indistinct. An emmetrope can
overcome this difficulty by substituting for the necessary accommodation a
convex glass of from 2 to 4 D, preferably behind the mirror. A myope lessens
his correcting glass by the same amount, so that myopes of less than 4 D
do best without any correcting glass, while a hypermetrope has to increase

the strength of his glass. It is well for the physician to emancipate himself from the need of this glass by prolonged practice.

Another trick is to have someone hold the tip of his finger at the place where the image must be formed, i.e., 7 cm in front of the lens; the observer fixes his eyes on the tip of the finger and maintains his accommodation when it is withdrawn.

A corollary to what has been said is that the observer must be certain in regard to his own refractive condition.
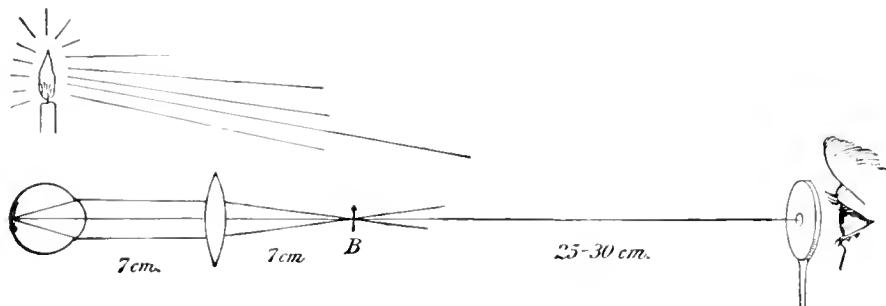


FIG. B.

## 4. The Question of Wearing Glasses

during an ophthalmoscopic examination has been answered by the above remarks. As the image lies in front of him at the distance of 25 to 30 cm, the ordinary reading distance, the physician wears the same glass that he uses to read with, particularly if he is presbyopic. The most convenient way is to place a glass of the proper strength behind the mirror.

## 5. Avoidance of Reflexes from the Lens and Cornea.

These reflexes can never be wholly avoided, even by the most expert. The student must learn to place them so that they do not fall directly on the place under observation. He may partially succeed in doing this by moving the lens a little to the right or left, so as not to look directly through its center, or by holding the lens in a slightly oblique position. Much is won when he learns to look past the reflexes, for the disturbance they cause is due not only to the fact that they cover the image, but also to the fact that they distract the attention and consequently excite a faulty accommodation in the eye of the observer.

Sometimes the reflexes come from other sources of light; this may be guarded against by seeing that the only light in the room is the one used for ophthalmoscopy, and that this is placed obliquely behind the patient, so that the eye to be examined is altogether in the shade.

Finally, the lens itself must be perfectly clean, for a dirty lens increases enormously the reflexes that appear.

## 6. Indistinctness of the Image.

After sight has been caught of the papilla it often appears to be very indistinct. This may be due to a variety of causes.

| *Cause* | *Remedy* |
|---|---|
| 1. The physician or the patient may have high degree of astigmatism, or of some other error of refraction. | He must therefore know the refraction of both himself and the patient, and may need to correct it. |
| 2. The distance may be incorrect. | Given under 2. |
| 3. His accommodation may be faulty. | Given under 3. |
| 4. Opacities may be present in the refractive media. | Therefore he throws light into the eye without using a lens before trying to see the fundus. |
| 5. The indistinctness may be due to disease of the optic nerve, retina, etc. | The beginner should not make this diagnosis until he is positive that the cause lies neither in himself nor in his technique. |

## 7. Incompleteness of the Image.

If only a portion, but not the whole, of the papilla comes into view, the examiner moves himself toward the side which he wishes to see, while the patient maintains his line of regard. For example, if only the left side of the papilla can be seen from the standpoint of the observer, the latter moves his head very slightly to the right. Of course the same result could be obtained by having the patient look a little more to the left, but the observer usually has his own movements under better control than those of the patient.

*If, in spite of all this, the observer has not succeeded in seeing the papilla, or any portion of it, it is best for him to break off the examination, to redirect the patient how to look* (see under 1), *and to try again.*

## 8. The Investigation of the Macula is often difficult, even for the expert, so there should be no hesitation to dilate the pupil when it cannot be seen clearly, and to examine the eye when in a condition of mydriasis. In doing this the rules should be observed that are given on page 11.

The macula is so placed that in order to bring it into view either the patient must look at the aperture in the center of the mirror, or the physician must move his own eye into the line of vision of the patient, while the latter remains looking in the same direction as during the presentation of the papilla. It is also possible, while the eyes retain their relative positions, for the physician to move the lens toward the patient's nose until the macula appears in its temporal margin.

**9. For the Examination of the Periphery,** which must never be omitted, the patient is told to look up, down, to the right, and to the left. The fundus can be seen in this way to within 5 mm of the ciliary body.

## II.  UPRIGHT  IMAGE

The difficulties in the way of an examination of the upright image lie in the relaxation of the accommodation and the management of the light. The following method seems the best to me, taking for an example an examination of the left eye. When practicable a drop of a 3 per cent. solution of cocaine, or of a 1 per cent. solution of homatropine, should be placed in the eye half an hour before the examination.

R Cocain. hydrochlorat ...... 0.3   R Homatropin. hydrobrom. ... 0.1
    Aquæ destil. ............ad 10.0     Aquæ destil. ............ad 10.0
      M. Sig.  One drop in the eye.      M. Sig.  One drop in the eye.

### Examination of the Left Eye

The light should be placed near the left side of the patient on a level with his eye. The physician and the patient should be seated on a level, and about 30 cm apart. The patient should be told to fix his eyes on a point on the wall situated in the prolongation of a line connecting his left eye with the left eye of the physician. The latter places his left hand on the right shoulder of the patient, or rather somewhat about the neck, so that he can direct the movements of the head as desired, then takes the ophthal-



FIG. C.

moscope in his right hand, interposes the glass that corrects his own refractive error plus that of the patient, approximately at least, holds it before his left eye, throws the light into the eye to be examined, and sees the red illumination of the pupil. Now he slowly approaches the patient, whose head he presses a little forward at the same time, ever keeping in view the red

illumination of the pupil and making it bright again, whenever it threatens to become indistinct, by little rotations of the mirror.

Thus the eye of the physician and that of the patient approach each other until they are only 1 cm apart. If the observer has not lost the light on the way, the papilla suddenly appears before him in its perfect beauty. If he has lost the light he draws quietly back from the patient and repeats the same maneuver. If it is lost again at the same place, the cause may be either an insufficient rotation of the mirror, a lock of hair, or the position of the light. He corrects the fault, whatever it may be, and begins again. Success is usually obtained after a few efforts. If the image is not quite distinct the lenses in the ophthalmoscope are slowly changed for stronger or weaker ones, while the observer watches until a sharply defined image of the papilla is obtained. The best way to do this is to look constantly at a certain blood vessel, and so to determine the correction. If the entire papilla is not seen, or if the physician wishes to look at another part of the fundus than that directly before him, he moves his head in the direction opposite to that in which the part lies which he wants to see. In other words, he looks at the fundus through the pupil just as he would look into a room through a keyhole; when he wishes to see the right side he moves his head to the left, and vice versa. The patient should continue throughout to look quietly in the same direction.

The right eye should always be examined with the right eye, the left with the left, and the light should always be on the same side of the patient as the eye that is being examined.

# DETERMINATION OF DIFFERENCES OF LEVEL IN THE FUNDUS

We perceive depth in ordinary life by means of binocular vision, but, as we can see with only one eye when we use the ophthalmoscope, this kind of perception of depth is out of the question. Still we can distinctly recognize differences of level in various ways.

1. By the so-called **Parallactic Displacement.** This is noted during the examination of the inverted image. If the convex lens is moved back and forth a little, the details of the fundus seem to move against one another if differences in level are present; the parts nearer the observer seem to be displaced more, or to move more rapidly, than those situated farther away. When a glaucomatous excavation, for example, is examined in this way the impression is given that its margins are slid forward over its base.

2. By the **Perspective Displacement.** When the observer moves a little from side to side during an examination of the upright image, the impression is given that the nearer places move in the opposite, while those farther away move in the same direction.

3. By the **Determination of the Refraction** of the parts that come into question. This also is done in the examination of the upright image. A hypermetropic eye is too short, in comparison with an emmetropic, i.e., its retina is nearer to the eye of the observer than that of an emmetropic eye, all other conditions being the same. Hence, when in an otherwise emmetropic eye a certain part, for example a choked disc, is greatly elevated so as to lie closer to the eye of the observer, such a part will be hypermetropic and form a contrast with its emmetropic surroundings. If the eye is myopic the elevated part will be less so than the rest of the fundus. In every case the elevated part has a lower degree of refraction than those portions that lie farther back, and vice versa. A difference in level can be calculated in millimeters from the difference in refraction, for a difference of refraction of 3 D corresponds to an elevation or depression of 1 mm. For example, if the retina of an eye is emmetropic and the papilla is hypermetropic 3 D, we know that the papilla is raised 1 mm above the retina.

4. When the difference of level is very great, as is the case in a bullous detachment of the retina, the different parts of the fundus can be seen at varying distances. It has been mentioned that in high hypermetropia, as in aphakia, we have to lean far back in order to see the fundus distinctly by the indirect method. This is the case to a much greater degree with the detached portion of the retina; while the parts that are not detached can be seen at the normal distance, we have to lean very far back in order to see those that are detached.

5. Great differences of level, as in detachment of the retina, can often be perceived better by simple illumination than by either the direct or the indirect method. Light is thrown into the eye, and then the normal portions appear to be bright while the detached parts are dark, or perhaps the detached bulla can be distinctly seen, especially if the observer draws rather near to the eye.

We indicate the **Place and Size of a Lesion in the Fundus** by reference to the papilla and its diameter, which is 1.5 mm. For example, we say that the size of a lesion is ½ a papillary diameter, i.e., that its diameter is 0.75 mm, or that it lies 2 papillary diameters from the temporal margin of the disc of the optic nerve, i.e., 3 mm distant.

# THE DILATATION OF THE PUPIL FOR THE PURPOSE OF AN OPHTHALMOSCOPIC EXAMINATION

is, under certain precautions, an absolutely harmless procedure, and it is to be recommended whenever the examination is rendered difficult by a small pupil. It is better to make an exact diagnosis with a dilated pupil than to make an incorrect or incomplete one because the pupil is too small. It is no confession of incompetence or ignorance. It is often almost essential for

the use of the direct method, or the examination of the macula. The only thing necessary is that certain precautions be observed, and these are:

**1. Never** *use atropine* to dilate the pupils for this purpose, because it renders paretic not only the sphincter pupillæ, but also the accommodation for about 8 days, so that the patient is unable to read or write for a week. What that means is readily appreciated by a physician who has once instilled atropine into his own eyes by way of experiment.

**2.** Care must be exercised in the case of old people, and if there is any suspicion of glaucoma. In the latter case it is best not to use any mydriatic at all; it is often superfluous, as patients with glaucoma usually have pupils that are somewhat dilated and react badly to light. Not more than one drop of the mydriatic should be placed in the eye of an old person.

## The most suitable mydriatics are:

R Homatropin. hydrobrom. . . . 0.1
    Aquæ destil. . . . . . . . . . . . ad 10.0
      M. Sig. One or two drops to
    dilate the pupil.

R Cocain. hydrochlor. . . . . . . . 0.3
    Aquæ destil. . . . . . . . . . . . ad 10.0
      M. Sig. One drop to dilate the
    pupil.

NOTE.—Not more than a single drop of cocaine should be used, because an exfoliation of the epithelium of the cornea may readily be induced by the application of many drops; and, while this is fairly harmless, it interferes with the view into the eye. Two, or even three, drops of homatropine may readily be used, except in cases of glaucoma and in old people. The method of instilling the drops is to draw down the lower lid with the forefinger of the left hand and to allow one drop of the solution to fall gently upon the inner surface of the lid from a dropper held in the right hand. It is of no use to instill a large number of drops, as they immediately escape. The patient is sent back into the waiting room and half an hour later we see if the pupil is dilated. Usually it is; if it is not, another drop is instilled and the eye is seen again 15 minutes later. The mydriasis begins after about 10 minutes and reaches its acme on the average in half an hour. Four or five hours later the pupil has usually regained its normal size. The accompanying disturbance of the accommodation is therefore comparatively slight, especially when cocaine is used.

# The Normal Papilla and the Normal Fundus

# Anatomical Review

A brief review of the anatomy is essential in order to understand the ophthalmoscopic picture of the papilla of the optic nerve. This nerve enters the eyeball through the lamina cribrosa of the sclera, to the inner side of and a little below the posterior end of the optic axis, and there forms the papilla.

The optic nerve is to be considered as a portion of the brain that has been projected forward, and, like the latter, it is enveloped in 3 sheaths, the dural, arachnoidal, and pial membranes, the interspaces of which correspond to those of the brain and are furthermore connected directly with the lateral ventricles. This fact explains how it is that an increase of pressure in the brain is transmitted into the optic nerve to produce a choked disc. The two outer sheaths pass over into the two outer layers of the sclera, while the inner one enters its innermost lamella, which forms the lamina cribrosa, and is connected with the chorioid. A number of vessels, which surround the optic nerve and are fed by the posterior short ciliary arteries, may be seen on transverse section in the neighborhood of the place where this change occurs. These vessels form *Zinn's*, or the *sclerotic, vascular plexus*. As this plexus gives off branches to the optic nerve a connection is formed between the vascular systems of the retina and the chorioid, but this union is of no practical importance.

Two segments, the anterior and the posterior, need to be differentiated in the intraorbital portion of the optic nerve, because of the vascular supply. The anterior segment is supplied by the central artery and vein of the retina, which enter it in the lower medial quadrant, 10 or 12 mm from the eyeball, and then run axially in the nerve. The artery comes from the trunk, or a branch, of the ophthalmic artery, which in turn is a branch of the internal carotid. The vein empties into the cavernous sinus, or into the superior facial vein, and has numerous anastomoses with other veins in the orbit.

The posterior segment receives its blood supply from a long, recurrent branch of the central artery of the retina and other branches of the ophthalmic artery, and discharges its blood into the cavernous sinus.

The **optic nerve** is circular on section in its orbital portion and is about 4 mm thick. It is composed of nerve fibers and connective tissue. The nerve fibers form bundles that run parallel to one another, and are interlaced together by an interchange of fibers, the number of which has been estimated

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at half a million. The nerve fibers have a medullary sheath, but no sheath of *Schwann*, and a supporting substance composed of neuroglia tissue lies between them. The pial sheath, which is closely adherent to the surface of the nerve, sends numerous trabeculæ and septa into it, where they join to form a network, and to envelope the bundles of nerve fibers. Within these are to be found the lymphatic and blood vessels.
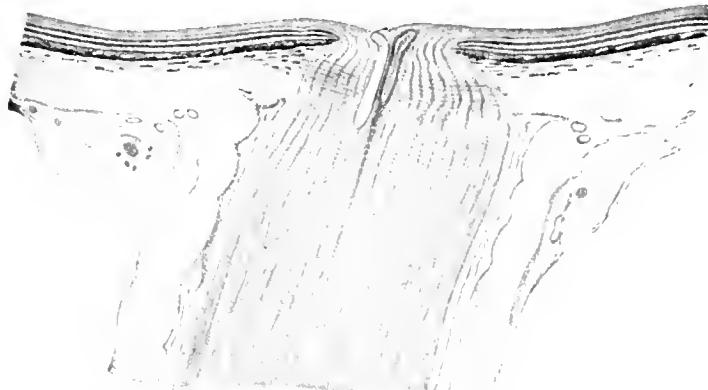


FIG. D.—Microscopic Section through a Normal Optic Nerve.

The lumina of vessels which form part of *Zinn's* vascular plexus can be seen at the place where the dural sheath bends over to join the sclera. The papilla is a flat surface in the drawing, with no marked excavation.

The caliber of the fibers of the optic nerve varies; the average is about 2 μ. The smallest are those of the papillomacular bundle, which supply the macula and form the medium of the finest vision. This bundle is of a longitudinally oval form, situated in the center of the nerve at the optic foramen, from which point it gradually approaches its temporal side, until at the place of entrance of the central vessels it lies wholly in its temporal margin and occupies the lower, outer sector of the disk in the form of a wedge with its apex inward.

At the level of the inner surface of the sclera are numerous fibers of connective tissue cutting transversely through the nerve, which, together with other fibers of connective tissue from the sclera and chorioid, form the **lamina cribrosa,** through the numerous meshes of which passes the optic nerve, which at this point is 1.6 mm thick. The diminution of its diameter is due to its loss of the medullary sheaths that are retained as far as the posterior third of the sclera, and are lost just before the nerve reaches the lamina cribrosa. This loss not only diminishes the caliber, but also causes a change in its color; while the medullated fibers appear white, the nonmedullated look rather grayish.

The fibers of the optic nerve bend outward in the papilla and are distributed in the layer of nerve fibers of the retina (see page 112).

# A. THE OPHTHALMOSCOPIC PICTURE OF THE NORMAL PAPILLA

The following points have to be noted, one after another, in a systematic examination:

1. Form and size.
2. Color.
3. Margins.
4. Conditions of level, excavation or protrusion.
5. Vessels.

1. The **Form** of the normal papilla is usually round, or slightly oval vertically. Less often it appears to be horizontally, or obliquely oval, a peculiarity which does not usually correspond to an actual anatomical condition, but is produced by an astigmatism of the cornea.

The variations in size are also only apparent as a rule: in hypermetropia the papilla **seems** to be larger, in myopia smaller, when examined by the indirect method, the reverse when seen by the direct. At the same time true differences in size are met with: sometimes the papilla is unusually small in the "little" hypermetropic eyes (see under Pseudoneuritis).

Attention may be called here to a mistake often made by beginners, who sometimes include a circular staphyloma, in congenital myopia, with the nerve and are led to think that the papilla is enlarged.

The color of the papilla is a delicate red which might aptly be compared to that of a peach blossom. The temporal side (upright image) is usually a little brighter than the nasal. In a large number of cases a specially bright spot is to be seen in, or a little to one side of, the center, which corresponds to the excavation about to be described.

2. The **Color** results from the combination of that of the lamina cribrosa and its meshes with that of the almost transparent fibers of the optic nerve. The former is almost white, except for the apertures, which have a gray appearance, while the optic nerve fibers, which are slightly gray, seem reddish from the presence in them of numerous capillaries. The observer sees through the almost transparent fibers to the lamina. At the places where the fibers are particularly well developed and densely packed, for example at the nasal margin in the upright image, the papilla appears redder than where they are less in number, as at the temporal margin, over which pass the few and delicate fibers that supply the macula (see Papillomacular bundle).

The brighter color of the temporal side is not, therefore, an indication of atrophy: this is indicated by a true white color.

On the contrary, those places in which the optic nerve fibers are almost wholly wanting, as at the bottom of an excavation, must normally be white, the color of the lamina cribrosa.

The color of the papilla is also influenced by its environment. If this

is very dark, as in brunettes, the optic nerve will seem to be particularly bright from contrast, and, on the other hand, it looks redder when the fundus is particularly pale.

The nature of the light, whether gas or electric, likewise exerts a certain influence, as the papilla appears to be paler or redder in proportion to the number of red rays it contains.

It is also affected by age, as in youth the red prevails strongly, while a yellowish tone is apt to be acquired in old age. According to *Elschnig*, the size of the papilla, which is not always constant, has an influence on its color. As it must be supposed that there cannot be any excessive difference in the number of nerve fibers that reach the normal eye, small papillæ are generally redder than large ones, because of the relatively denser layer of bundles of nerve fibers with the capillaries between them.

3. The **Margins** of the normal papilla are sharply defined. Special marginal rings are frequently present, one white, the so-called connective tissue ring, or scleral ring, and one black, the so-called pigment ring, which is sometimes termed erroneously the chorioidal ring.

The white ring may be due to two different anatomical conditions: it may be either a true connective tissue ring, separating the chorioid from the sheath of the optic nerve, or the sclera itself covered with rudimentary chorioid and marginal tissue (see Fig. **1**).

The black ring is produced when the pigment layer of the retina in the neighborhood of the optic nerve is particularly thick and this thickening stands out prominently. If this ring lies close to the papilla a connective tissue ring cannot be seen, but otherwise there may be seen first a connective tissue ring and then a ring of pigment. It is only in exceptional cases that these circles are complete; segments only are visible, as a rule, and these are usually on the temporal side; frequently there is an accumulation of pigment instead of a black line, and sometimes one or both of the rings are entirely absent.

Sometimes the chorioid, retina and sclera are pushed over the nasal margin of the papilla, so that this appears thick and indistinct (see Fig. I in the text and Fig. **5**). This happens more often in myopic than in emmetropic eyes. The margin of the papilla, that is, the portion of the optic nerve that is covered, shines quite weakly through the tissue as a yellow crescent, the supertraction crescent (see Fig. I in the text). The margin is more distinct on the temporal side than on the others for the reasons already mentioned.

Age again plays a certain part, for the zone of pigment is usually developed considerably more in infants than in adults.

## 4. Excavation of the Papilla.

The name papilla dates back to the time when it was thought to be an elevation at the entrance of the optic nerve. This was an erroneous anatomical

idea; the papilla rises above the level of the surrounding retina only in exceptional cases, as a rule it is of the same height.

Two types need to be differentiated:

(*a*) The flat papilla, in which the spreading out of the fibers of the optic nerve takes place wholly on a level with the retina. The color of such a papilla is almost uniformly reddish, there is scarcely any difference in color between the nasal and temporal portions, and the white spot, which indicates the excavation in the other type, is nearly absent (see Fig. D in the text).



Fig. E.—Small Excavation in the Temporal Part of the Papilla.
The margins of the papilla are slightly elevated and surround a funnelshaped excavation.

(*b*) The excavated papilla. The excavation is due to the fact that the fibers of the optic nerve do not completely fill out the hole in the chorioid; they cling to the wall of the sclerotico-chorioidal canal and leave in the center, as they swell out like a fountain, a larger or smaller funnelshaped or cupshaped cavity below the level of the surrounding tissue. The presence of this cup is recognized ophthalmoscopically from the fact that the reddish color changes either suddenly or gradually to a whitish, or to white.

The size of the excavation varies a great deal. It may occupy only a very small part of the papilla, or it may be so large as to reduce the normally colored portion to a narrow circle or crescent, but a colored zone always lies between it and the adjacent retina (see Fig. N in the text and Fig. **15**).

The transition from the tissue proper of the papilla to the excavation may be either gradual or abrupt. This is to be perceived from the behavior of the vessels; in the former case they pass without visible bending into the white place, in the latter they suddenly bend like hooks. While they may be seen clearly and distinctly from the margin of the papilla to that of the excavation, they suddenly become indistinct at this point and appear as bright, indistinct bands at the bottom of the excavation, or become more or less invisible. If they plunge downward very abruptly they have a markedly dark color at the edge of the excavation, and simulate there a very dark

swelling. Hence it is that the light streak, visible everywhere else, is absent at the place where the vessel bends (see page 105 and later); perhaps also because at this place the vessel is seen, as it were, end on, as it plunges downward.

The reason why the vessels in the floor of the excavation are sometimes seen either not at all, or to follow different courses, is that they do not
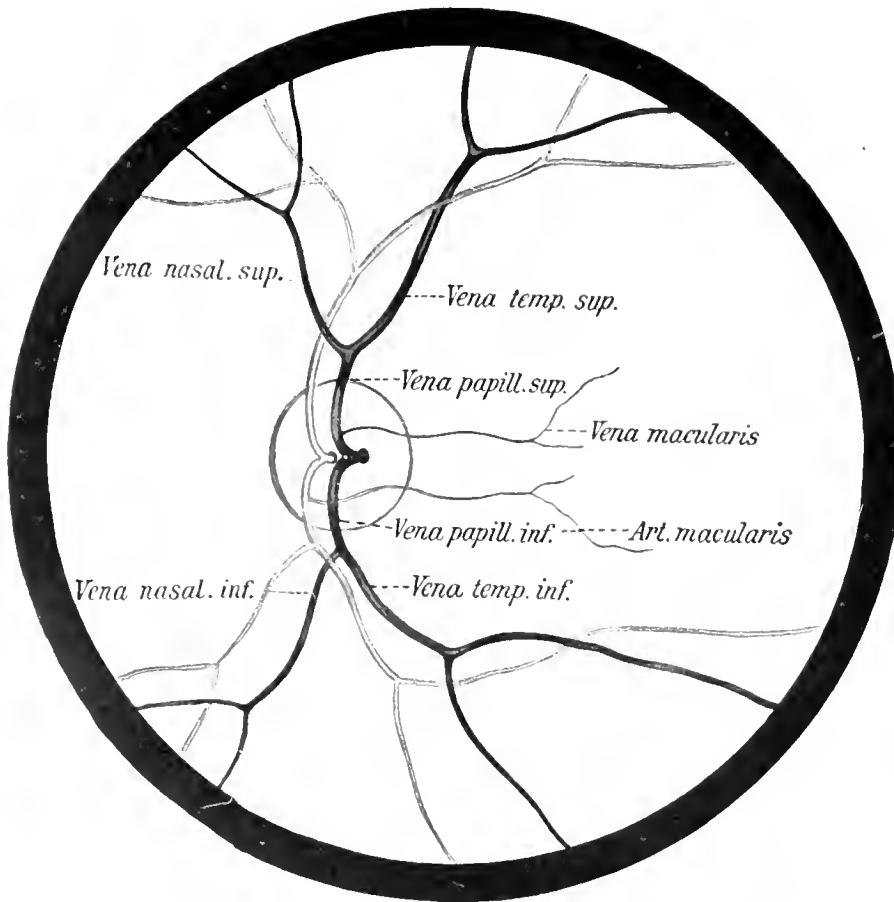


FIG. F.—Schematic Drawing of the Fundus, Upright Image.

plunge straight down from the margin of the excavation, but bend to one side: in this case the apparent end of the vessel is apt to be pointed, like the beak of a bird (see Fig. M).

The excavation may be 1 mm deep. As 1 mm = **3 D** (see page 10), a change must be made in the lenses of the ophthalmoscope in order to bring into focus the vessels at its bottom when using the direct method. If both the observer and the patient are emmetropic, a —3 D lens must be inter-

posed to enable the former to see them clearly. Parallactic displacements also are visible when the depth is sufficient.

The position of the excavation is usually central; it frequently extends into the temporal portion, rarely into the lower. Sometimes it reaches to the
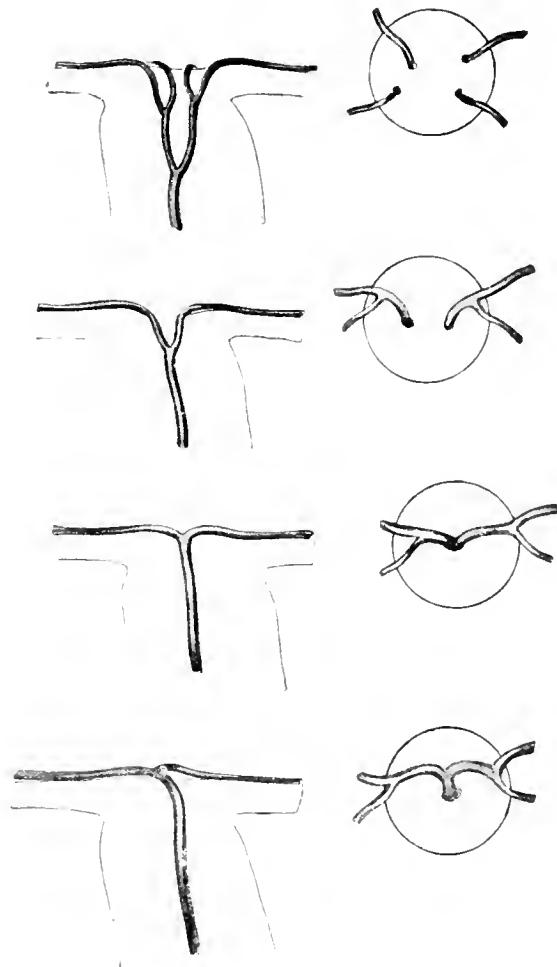


Fig. G.— (Schematic.)

This figure is intended to show in what way the great variety in the courses of the retinal vessels comes to take place. If the vessel divides within the optic nerve it is not the trunk, but the branches that emerge from the papilla.

temporal margin, but there it slopes away gradually. *Every abrupt excavation that extends to the margin of the papilla is to be considered pathological.*

The bottom of the excavation is not uniformly white; usually the central portion alone is of this color, while the peripheral parts have a reddish gray tone. Dark points may also be seen in the white itself; these are caused by the meshes of the lamina cribrosa.

## 5. The Vessels.

The artery and vein divide, to draw a very schematic picture, sending a branch upward and another downward, the superior and inferior artery and vein of the papilla, and each of these subdivide into two branches, the superior temporal and nasal arteries and veins and the inferior temporal and nasal arteries and veins. Each of these divide again into two branches, from which in turn spring two smaller ones, and so on. No anastomoses are present. But such a regular subdivision as this schematic drawing is actually seen only in extremely rare cases—at least, I have not seen a single one in the many thousands of eyes that I have examined ophthalmoscopically for the purpose of reproduction in this atlas—for the subdivisions are not symmetrical. Sometimes the vessel divides within the optic nerve, and then we see not the trunk, but the two principal branches coming out of the papilla; perhaps one of these has already divided so that apparently 3 vessels emerge from the papilla. In at least the great majority of cases the 4 chief branches can be differentiated as such. It is a matter of importance to know this fact, because it may happen in certain diseases that the vessels atrophy until they are invisible, so that only from their entire number can the absence of a vessel be disclosed.

Attempts have been made to utilize this condition, which is not regular, but individual to every man, according to *Bertillon's* system for the purpose of identification, but they do not seem to have been successful as yet.

The vessels of the macula come, as a rule, from the superior and inferior temporal arteries and veins, which are usually larger than the corresponding nasal branches, but they frequently arise directly from the common trunk, and in many cases from the system of ciliary vessels (see under Cilioretinal vessels).

The arteries and veins can be distinguished apart easily.

The arteries are more slender than the veins, are of only about two thirds the size, follow more direct courses, and are sharper in their outline. They are bright red and have distinct reflexes, the breadth of which is about one quarter the diameter of the vessel. The veins are wine red and have light streaks which are considerably narrower, one fourteenth of the diameter of the vessel, and are by no means so distinct as those on the arteries. This light reflex probably comes from the surface of the blood column, rather than from that of the vessel itself. The wall of the vessel is perfectly transparent and is perceptible only under pathological conditions.

**Arteries** and **veins** frequently cross, but no regularity can be observed as to which of the two is the upper and which the lower, although wherever two vessels cross one is always an artery and the other a vein. Branches of the same kind of vessel never cross, a fact that can often be utilized to determine the nature of a vessel which cannot be seen distinctly.

## Venous Pulse.

No pulsation can be seen in the arteries under normal conditions, while, on the contrary, the venous pulse is a phenomenon that can be observed in most men, though it is ordinarily not very marked. It is to be seen only in the principal venous trunks which lie on the papilla, and appears there most distinctly in the veins that are flattened to the greatest degree, or seem to end in a point on the papilla, or in those that bend at a right angle and form a dark knee at the place where they bend. In an abrupt excavation of the optic nerve the pulsation can be seen best in the veins that bend over the margin of the cup at an acute angle. Only one venous trunk pulsates, as a rule. The contraction and paleness of the vein begins just before the beat of the radial pulse, extends from the center toward the periphery, but rarely passes over the margin of the papilla. Immediately after the radial pulse comes the dilatation and filling of the vessel from the periphery toward the center.

The way in which the venous pulse is brought about is explained in a variety of ways. One theory is that it is a negative pulse caused by the activity of the right ventricle and auricle of the heart; another is that it is caused by the continuous transmission of the pulse wave from the arteries through the capillaries into the veins, the possibility of such an occurrence being provided for by the comparatively high extravascular, i.e., intraocular, pressure. A third theory is that an elevation of the intraocular pressure is created by the arterial wave of blood, which compresses the soft-walled veins, and then, at the instant when the intraocular tension falls, synchronously with the cessation of the *arterial* wave of blood, the veins refill.

A spontaneous arterial pulse must always be considered pathological.

If the venous pulse is not visible otherwise, it can be produced by making a slight pressure on the eyeball. Greater pressure may excite a lively arterial pulse.

**Vascular anomalies** which are to be looked upon as normal are:

1. *The Cilioretinal Vessels.*

These emerge in the form of hooks at the periphery of the papilla, or in the region of the connective tissue ring, and pass into the retina like the other vessels. They arise from the ciliary arteries, *Zinn's* vascular plexus, and thence have derived the name of cilioretinal vessels. They are almost all arteries: it is exceptional to meet with a vein. According to *Elschnig* they can be seen in every seventh eye.

2. *The Opticociliary Vessels.*

These are branches of the central vessels which pass into the vascular system of the chorioid without touching the retina. They are very rarely to be found in normal eyes but are more common, as newly formed vessels, in pathological conditions, such as glaucoma, choked disk, and wounds.

# B. THE FUNDUS OCULI.

The **color of the fundus** is greatly influenced by the color and density of the pigment; the color of the blood vessels in the chorioid is of less importance. The visual purple can in no way exert any influence, although its
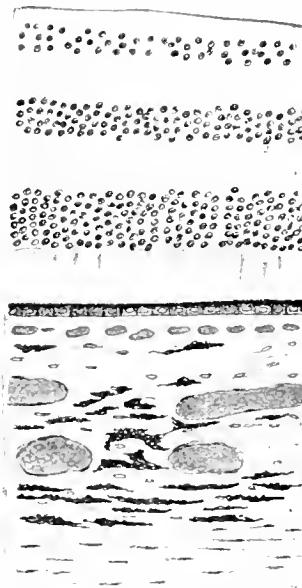


Fig. II.—Distribution of Pigment.

The retina (above) terminates next to the chorioid with the layer of pigment epithelium. If this layer is very dense nothing can be seen of the chorioid, and we have the uniform, stippled fundus. In the chorioid the pigment is situated chiefly between the vessels; if the layer of pigment epithelium is not very dense the chorioidal pigment can be seen, and we have a tesselated fundus. If the chorioidal pigment is also wanting the fundus is albinotic.

name might lead one to suppose it could, for it is found only in eyes adapted to the dark and is transformed in the light into "visual white."

*Pigment* (see Fig. II) is found

(*a*) in the layer of pigment epithelium of the retina, and

(*b*) in the intervascular spaces of the chorioid.

We distinguish, according to the quantity and distribution of the pigment in these two membranes,

## Three Types of the Normal Fundus.

1. The *Uniform, Stippled Fundus* (Fig. **1**).

The uniform appearance of this type of fundus is brought about by the fact that the layer of pigment epithelium contains so much and such dense pigment that the chorioid beneath it is completely hidden from the eye of the observer. The tone of color is red, brown red, or black brown, according to the quantity of pigment.

2. The *Tesselated Fundus* (Fig. **2**).

In this type the layer of pigment epithelium contains less coloring matter; consequently it is possible to see through the almost transparent retina and to perceive the markings of the chorioid. The reddish chorioidal vessels are seen to form numerous anastomoses, and the pigment of the chorioid is massed in the intervascular spaces between them. The vessels appear as bright bands on a dark background.

3. The *Albinotic Fundus* (Fig. **3**).

In this type the layer of pigment epithelium contains little or no pigment, so the markings of the chorioid are again visible. But this membrane also has no pigment, and consequently the sclera is seen to shine through the retina and chorioid, forming a yellowish white background, upon which the chorioidal vessels appear as dark bands. They can be distinguished from the retinal vessels by the absence of the light reflex, their abundant anastomoses, and their deeper position.

Cases are often met with which do not belong exclusively to any one type, but present the characteristics of two or more. The layer of pigment epithelium may be thick enough to hide the markings of the chorioid in some places, while in others it is thinner and allows the chorioidal vessels to appear on a dark (Type II), or a bright background (Type III).

The pigmentation is usually densest about the papilla and in the region of the macula, so that even in an albinotic fundus the chorioidal vessels are not usually visible in the macula, although they can be seen in the less pigmented places in the periphery.

The abundance of pigment in the fundus is usually in keeping with that in the hair and skin of the individual, so that we speak of a blonde, and of a brunette fundus.

The retinal vessels can easily be distinguished from the chorioidal, even in the albinotic eye, by noting the following characteristics:

| Retinal Vessels | Chorioidal Vessels |
| --- | --- |
| appear to be round, | appear to be flat, |
| have light streaks, | have no light streaks, |
| divide dichotomously, | divide irregularly, |
| form no anastomoses, | form many anastomoses, |
| converge toward the papilla, | have no uniform direction, or converge toward the periphery (vortex veins). |
| are superficial. | are deep. |

The **Course** of the **Retinal Vessels** varies according to the refraction of the eye. In myopia they are markedly drawn out, while in hypermetropia a marked tortuosity, especially of the veins, can often be seen. This tortuosity is due to the growth of the eyeball being too little as compared with

the design of the vessels. The normal conditions of circulation (for pressure pulse see page 107) and the absence of any morbid symptoms differentiate this from other forms of tortuosity, which are caused by morbid conditions of the vessels and inflammations.

The **Retinal Reflexes** form a very marked phenomenon, especially in young persons, as they appear chiefly along the vessels and in the region of the macula (see Fig. 2). They appear in the forms of bandlike, or island-like spots, which can be recognized easily to be reflexes by the fact that they change their forms and positions with movements of the head and mirror. They are particularly distinct when the vision is focussed on the deepest part of the vitreous, as when a +1, or a +2 glass is used in looking at the fundus of an emmetropic eye; they are less distinct when the pupil is dilated than when it is contracted. The explanation of these reflexes is that, in consequence of the elevation of the surface of the retina by the vessels, concave grooves are formed which act like concave mirrors. A bright curved line can be seen in many cases on the nasal side of the papilla, running parallel to its margin at the distance of about one papillary diameter. This is known as *Weiss'* reflex ring. It was thought by its discoverer to indicate a detachment of the vitreous, and to be pathognomonic of myopia, but this theory cannot be correct, as the line is met with in emmetropia and hypermetropia. It must not be confounded with the *Weiss-Otto* shadow ring, which is met with in high myopia and indicates the margin of a sclerectasia, the so-called staphyloma verum (see Fig. **73**).

The **Chorioidal Vessels** have been described already, so it will suffice to say that they gather the blood into large veins, the vortex veins, which, to the number of four or more, usually lie in the periphery. In exceptional cases, oftenest in myopia, they lie at the posterior pole, as shown in Fig. **3**.

**Macula.** The macula deserves a special description. This portion of the fundus oculi has been termed the macula lutea because it contains a yellow coloring matter; yet the area that contains this yellow coloring matter is considerably larger than the place that is designated ophthalmoscopically by this name. It may be recognized from the behavior of the blood vessels, which surround and direct their points at it without reaching it. The area thus surrounded by, but lacking in blood vessels, lies about $1\frac{1}{2}$ papillary diameters from and a little above the papilla. It has the form of an oval, 5 papillary diameters (P. D.) broad, and $2\frac{1}{2}$ P. D. high. Its center appears rather dark. This, the macula lutea in the narrow sense, is surrounded in young people by a brilliant reflex ring $2\frac{1}{2}$ P. D. broad and 1 P. D. high. This ring surrounds the part of the macula lutea which contains no nerve fibers. In the center of this ring the so-called reflex of the fovea can usually be seen in children, caused by the reflection of the light from the sides of the foveal funnel, and so it appears sometimes round, sometimes crescentic, sometimes wedgeshaped, according to the way in which the light is thrown and the mirror held.

# PLATE I

Fig. 1.  Normal Fundus of the Uniform, Stippled Type

Fig. 2.  Normal Fundus of the Tesselated Type, with Numerous
Reflexes from the Retina

# Fig. 1.   Normal Fundus of the Uniform, Stippled Type

If we study the papilla in the way repeatedly mentioned in the text, re-garding in turn its form, its margins, its differences of level, and its vessels, we see the following details: The papilla is vertically oval, has sharply de-fined margins, very clearly marked connective tissue and pigment rings, and a shallow excavation in its center. It is normal in color, the temporal por-tion distinctly brighter than the nasal. The pigment epithelium is so dense that no details of the chorioid beneath it can be perceived. The pigment is particularly concentrated about the papilla and in the region of the macula. The dark, larger vessels, without distinct light streaks, are the veins; the brighter, narrower ones, with distinct light streaks, are the arteries. Al-though their subdivision is not quite regular, yet the division above and below of both the arteries and the veins into 2 principal branches can be seen. A small branch of the artery and of the vein approaches the macula.


# Fig. 2.   Normal Fundus of the Tesselated Type, with Numerous Reflexes from the Retina

In contrast to Fig. 1 the markings of the chorioid can be seen over the greater part of this fundus. This is because the pigment layer of the retina is very thin and allows the tissue beneath it to show through. The dark, islandlike places are formed by the pigment of the chorioid that lies between its vessels (intervascular spaces, see page 25). As the vessels of the chorioid are brighter than the pigment lying in their vicinity, we say that the tes-selated fundus is characterized by bright chorioidal vessels on a dark back-ground. The papilla is vertically oval, and has a distinct connective tissue ring, but no pigment ring. Its temporal portion is brighter than its nasal. The pigment of both the retina and the chorioid is lacking in its vicinity, so there is a bright zone about the papilla. The macula and fovea can be seen quite distinctly because the reflections at their margins and along the courses of the vessels are very great. The bright spots in the vicinity of the macula can be recognized to be reflections from the fact that they change whenever the mirror is moved. This picture shows that there is little pig-ment in the retina, but plenty in the chorioid.

Tab. I.
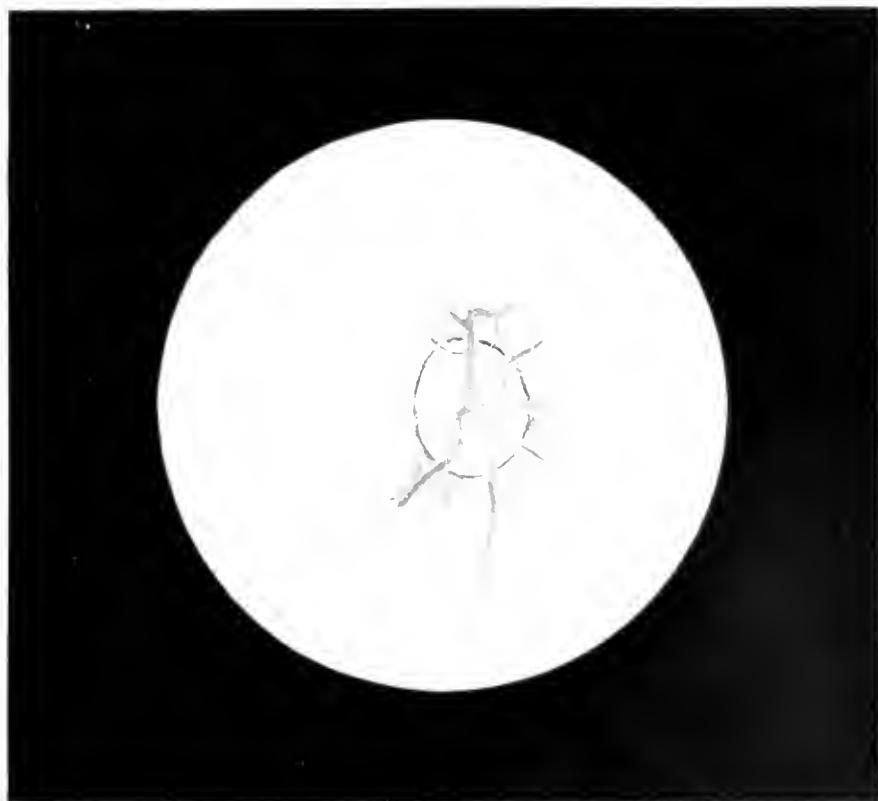


Fig. 1.
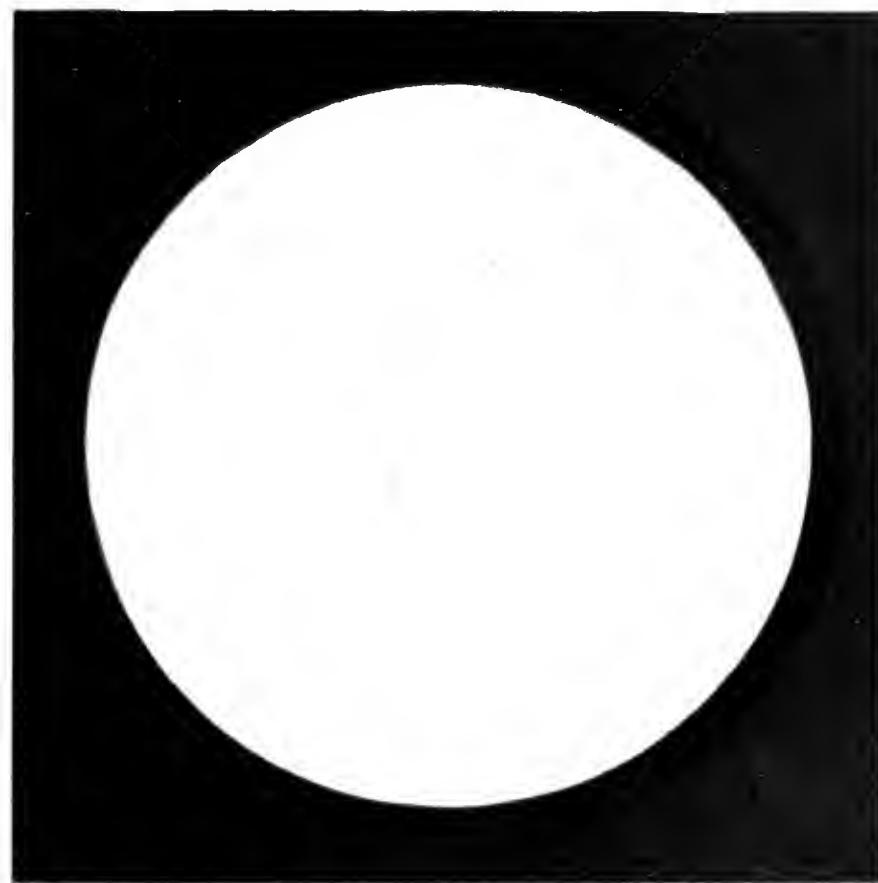


Fig. 2

PLATE II

Fig. 3. Albinotic Fundus

## Fig. 3.  Albinotic Fundus

This picture shows a complete absence of pigment in both the retina and chorioid.  The vessels of the latter can be very plainly seen to unite into larger trunks, the vortex veins.  The arteries and veins cannot be distinguished from each other.  The conflaence of the vessels of the chorioid in the neighborhood of the papilla is rather unusual, in most cases this takes place in the periphery, in the region of the equator.  The complete absence of pigment in the region of the macula is likewise not common; more often there is a distinct accumulation of pigment at this place, even when the albinism is perfect otherwise.  The papilla is bright red, but only in consequence of the effect of contrast with its pale surroundings, its margins are sharply defined, the excavation is very shallow, the retinal vessels are normal.

For the differentiation between the vessels of the retina and those of the chorioid, see page 25.

A partially albinotic fundus is shown in Fig. **8.**

Tab. **2.**
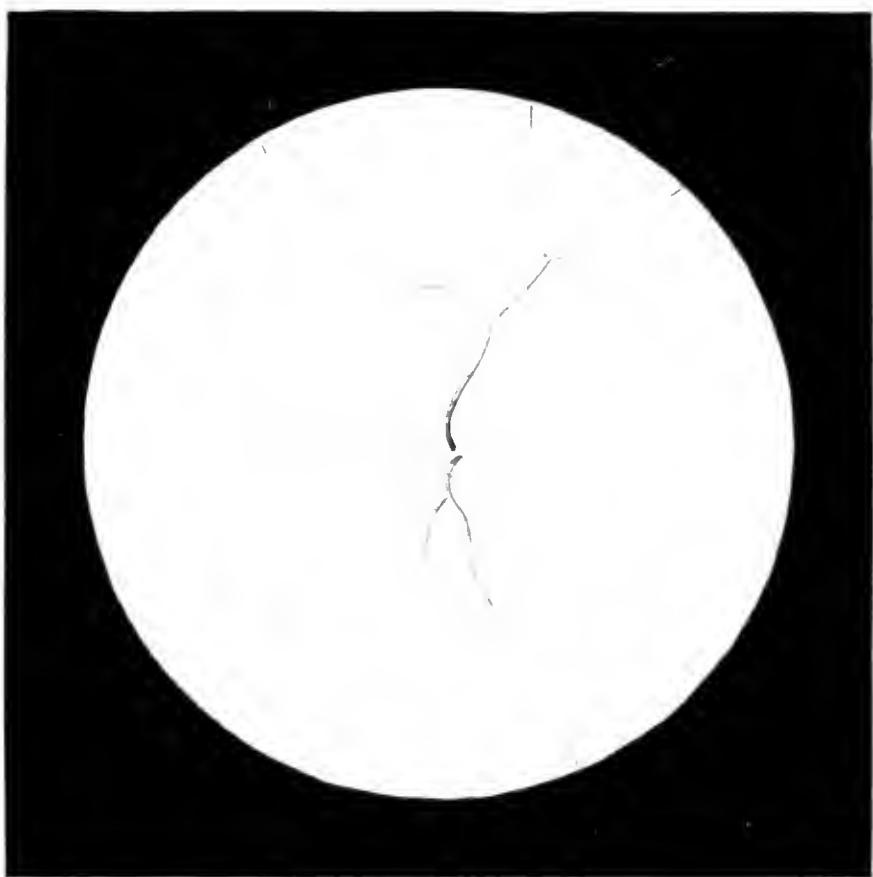


Fig. 3.

# Conus and Staphyloma

## Differential Diagnosis of the White Rings and Crescents to be Found in the Immediate Vicinity of the Optic Nerve

# Conus and Staphyloma

## Differential Diagnosis of the White Rings and Crescents to be Found in the Immediate Vicinity of the Optic Nerve

The head of the optic nerve affords a number of anomalies of this nature which are apt to be thought parts of the papilla by those who are not expert, and to be grouped by them under the term "large papilla," but, with some attention and knowledge to the factors that enter into the problem, it is not difficult to differentiate the individual conditions, and to draw from them important conclusions with regard to the diagnosis.

The principal conditions to be taken into account are:

1. Conus temporalis.
2. Staphyloma posticum.
3. Peripapillary atrophy of the chorioid.
4. Conus inferior.
5. Halo.
6. Medullated nerve fibers.

I exclude here all inflammatory affections, such as optic neuritis, great fullness of the vessels, œdema, and hemorrhage, and emphasize the point that this classification serves a purely practical purpose. Conditions are grouped together which have nothing to do with one another, either anatomically or etiologically, but have only some features in common; they have a certain resemblance to one another in form, color, and position, which may lead those who are inexperienced into error, and, on the other hand, one of them may appear alone in an otherwise normal fundus.[1]

The features common to all these conditions are:

1, the color, which is usually a yellowish, or bluish white;
2, the position, in the immediate neighborhood of the papilla;
3, the form, of which the crescent and the circle are the principal types;
4, the absence of signs of inflammation.

In order to differentiate the individual conditions we will group them first with regard to their positions as respects the papilla, as they are seen in

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[1] A number of other conditions might be included, such as coloboma of the sheath of the optic nerve, certain forms of coloboma of the chorioid, abnormal development of the glia tissue and of connective tissue, but they have been omitted because of their rarity.

the upright image, remembering that everything is reversed in the inverted image.

(a) On its *temporal side* lie, or may lie
>> the conus temporalis,
>> the staphyloma posticum,
>> the peripapillary atrophy.

(b) *Below* it
>> the conus inferior,
>> medullated nerve fibers.

(c) *Surrounding* it
>> the halo,
>> the staphyloma posticum annulare,
>> the peripapillary atrophy.

(d) *Above* it
>> medullated nerve fibers, or
>> the peripapillary atrophy.

Such an arrangement as this may seem at first glance to be rather risky, because other conditions may combine with those that have been mentioned and overthrow the artificial fabric; thus the staphyloma posticum may appear in company with hemorrhages, or with diseases of the macula, or, in exceptional cases, retinitis albuminurica may present an appearance which seems at first sight similar to that of these conditions. But I consider this differential diagnosis to be of sufficient importance to introduce it at this place in spite of these objections.

1. The **Conus** (Figs. **4** and **5**) is a uniformly yellowish white crescent that ordinarily lies to the temporal side of the nerve, to the nasal side in the inverted image, which rarely attains at its widest part the breadth of half the diameter of the papilla. Toward the retina it usually presents a more or less broad edge of pigment. This is the common form of conus, which, when typical, can be distinguished from the staphyloma by the fact that it exhibits no visible vessels of the chorioid.

The conus inferior will be described a little later.

Certain deviations from this typical form are met with. The conus may, though rarely, lie on the opposite side of the nerve, but then it extends, at least partially, toward the temporal side. In rare cases it may surround the papilla, but then the temporal portion is the broadest.

The color may vary also. The crescent may be white only at the margin of the optic nerve and may have a reddish yellow, or a reddish brown tone toward the retina, or the entire crescent may be of such a color.

A change in the medial margin of the sheath of the optic nerve, the so-called supertraction, may be found comparatively often at the same time with the conus temporalis. This is due to the fact that the retina, chorioid, and it may be even the sclera, cover the optic nerve at this place, so that it can be seen only indistinctly through these membranes.

The conus may be the result of various anatomical conditions, which differ again as they are congenital or acquired.

*A.* The **congenital conus** is due to the fact that the outer layers of the retina and of the pigment epithelium, as well as the chorioid, are rudimentary, or not formed at all, over the area that is white, so that the sclera shows through. This form differs only quantitatively from the scleral ring.

It can readily be understood that this form of conus occurs in other than myopic eyes, and that it is not necessarily strongly marked on the temporal side.

*B.* **Acquired conus.** This is the result of the stretching that takes place in the posterior part of the globe in myopia. It may be caused in various ways, and it is necessary to recall the normal configuration of the
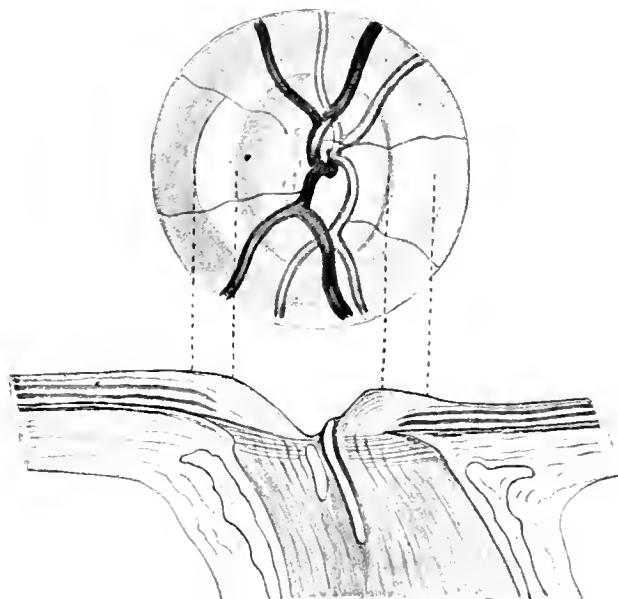


FIG. I.—Head of the Optic Nerve in Myopia, after *Fuchs.*

The upper drawing gives the ophthalmoscopic appearance presented by the condition delineated in the lower. When compared with Fig. D the oblique course of the optic nerve is striking; it does not pass through the sclera thus ╱......╲, but thus ╲......╲. Consequently the sclera is seen through the retina and chorioid on the temporal side, on the left side in the drawing, which gives the picture of a white crescent, or conus. On the opposite side the optic nerve is covered by the retina and chorioid, partly also by the sclera, so that this portion of it is seen only through these membranes and appears as an indistinct, ill-defined crescent, which is called the supertraction crescent.

sclerotic canal, the aperture in the sclera through which the optic nerve passes, in order to understand it. Normally this canal forms a funnel with its smaller opening forward, thus ╱....╲, but when a conus is present the temporal side has been ground off so as to form an oblique canal with parallel

walls, thus \\ . . . . \\. As the tissue covering the optic nerve is transparent the wall of the canal at this place can be seen, and forms the ophthalmoscopic picture of a conus, also called a *distraction crescent*.

Another form of conus, called the retraction crescent,[1] is produced in the following way:

The retina and the chorioid do not yield equally in the stretching at the posterior pole, the elastic lamina of the chorioid, in particular, not giving way in like manner as the retina. As it does not simply stop at the margin of the optic nerve, but is intimately united with its interstitial tissue, a fold of the optic nerve fibers is apt to be torn between the retina and the stroma of the chorioid. The latter then perishes, so the white color at this place is caused by the color of the sclera plus that of the glia fibers over it.

With regard to the origin of the so-called supertraction crescent, see Fig. I in the text and the accompanying explanation.

Aside from the rare cases of congenital conus that may be met with in hypermetropia and emmetropia, we must consider

## the Conus as a Sign of Myopia.

We may go even a step farther. There are two forms of myopia, the acquired and the congenital. The former develops during school life, especially in children in the higher grades, and near work is an etiologic factor in its production. This is the benign form of myopia, which rarely exceeds 6 or 7 D, and is complicated only in exceptional cases by diseases of the macula, or detachment of the retina. The pathological condition in this form is a *uniform* stretching of the segment of the eyeball that lies behind the equator. As this process of stretching is very slow, uniform, and of comparatively slight degree, and as it usually stops when the body ceases to grow, its effect is exhausted in the formation of the conus which has been mentioned.

It is otherwise with congenital [2] myopia. This ordinarily exhibits pathologically a *circumscribed* stretching, confined to the region of the posterior pole, but one which is much more marked than that in the form just described. It is associated with a greater or less degree of outward bulging of the sclera, and gives ophthalmoscopically the impression of a staphyloma posticum. This is the malignant form, which is apt to be complicated by

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[1] The nomenclature of these crescents is not uniform. Many writers call the one here named the retraction crescent, the distraction crescent, and vice versa. Others make no distinction between conus and staphyloma, understanding by the latter only a true bulging outward of the posterior pole of the eyeball, and by conus the ophthalmoscopic appearance caused by that condition, indifferent to whether it presents obliterated vessels or not.

[2] The expression "myopia to which the predisposition is congenital" is, perhaps, a better term than "congenital myopia," for one that develops spontaneously without any external provocation, like near work, must be taken into account.

changes in the macula and detachment of the retina, and to attain a high degree.

Between these two extremes are transitional forms, but in general it may be accepted as a fact that,

**the conus is an indication of acquired myopia, or "school myopia," and the staphyloma is an indication of congenital myopia.**

2. The **Staphyloma** (Figs. **6, 70**-**73**) appears ophthalmoscopically as a white crescent, usually larger than a conus, which is situated in most cases on the temporal side of the papilla. Less often it surrounds the nerve, but even then its broadest part is on the temporal side of the latter. In contrast to the condition presented by the conus, sclerosed chorioidal vessels are found either in the crescent, or in its immediate vicinity.

The name staphyloma deserves explanation. Properly speaking, the above-mentioned outward bulging of the posterior pole of the eye is to be understood when we speak of a staphyloma, but the term is also applied to the crescent, which is the ophthalmoscopically visible evidence of its presence.

Aside from an outward bulging and a thinning of the sclera, the same pathological conditions are to be found as in acquired conus, except that atrophy of the chorioid, particularly of its vessels, forms so prominent an addition to the symptoms that we may say, cum grano salis:

*The presence of sclerotic vessels of the chorioid, especially in the vicinity of the crescent, is indicative of staphyloma, their absence of conus.*

Still, the breadth of the crescent and the presence of other changes must be taken into account in making the diagnosis.

Fig. **6** shows a staphyloma posticum in the early stage of its development. The sharply defined crescent can be seen to contain chorioidal vessels, some of which are totally obliterated, while some contain blood, with the black pigment of the intervascular spaces distinctly visible between them. On the farther side of the margin of the staphyloma are to be seen chorioidal vessels that have undergone similar alterations, but these are still covered by the veil of pigment, which is absent over the area occupied by the crescent. This staphyloma may hasten through all the stages of atrophy of the chorioid until it is total, as shown in Fig. **54**. Sometimes the time of the completion of the atrophy varies in different parts of the staphyloma, so that breaks appear through which the pigment of the chorioid can still be perceived within it in some places, while in others it cannot (Fig. **70**). In many cases, however, these breaks are not caused by the unequal advance of the atrophy, but by shadows that are produced by irregular outward bulgings at the posterior pole. Such a shadow may also appear around the posterior pole, but it is usually to be seen only on its nasal side, and is frequently double, or multiple, giving rise to a terraced appearance (Fig. **73**). A fine, brilliant, reflex curved line is frequently to be seen on the nasal side of the papilla, which was once thought to indicate a collection of fluid between the retina and the chorioid, and a commencing detachment of the vitreous, but this interpreta-
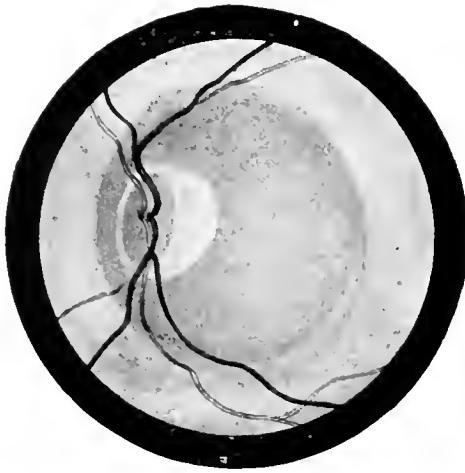
FIG. J.

Schematic sketch to show how the papilla is caused to appear out of drawing in high myopia. The ectasia affects exactly the posterior pole of the eye; the papilla is situated in its nasal wall and is consequently seen in half profile with its horizontal axis foreshortened. The white crescent, the staphyloma, is directed toward the center of the ectasia.
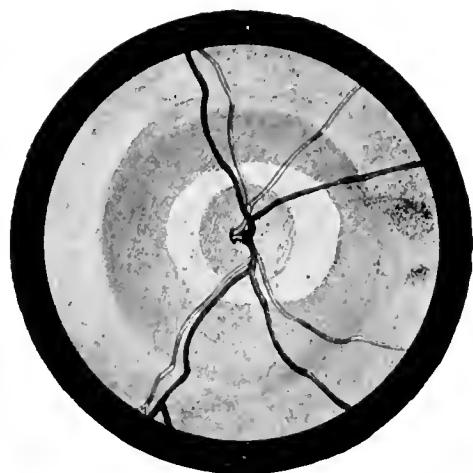
FIG. K.

In this case the ectasia does not lie exactly at the posterior pole but rather below it; hence the papilla is not situated exactly in its nasal wall, but in its upper and nasal, and the foreshortening is consequently of its oblique axis. The white crescent, the staphyloma, is directed toward the center of the ectasia.



FIG. L.

In this case the ectasia lies to the nasal side of the posterior pole so that the papilla occupies the bottom of its cavity. Consequently we look directly at it, and it appears to be of its natural, round form. The staphyloma is circular.

tion is not correct, as such a line sometimes appears in other conditions of the refraction (see page 26).

The papilla itself may appear either normal, or reddish and indistinct when a staphyloma is present. The redness and indistinctness are due partly to the pulling, partly to the effect of contrast with its bright surroundings. The retinal vessels are very slender and drawn out (see page 105). The form of the papilla is quite interesting. As a rule it is not round, but oval, with its short diameter vertical to the broadest part of the staphyloma; i.e., if the latter is broadest in the horizontal meridian the papilla appears to be vertically oval; if the broadest part inclines somewhat downward from this meridian, the principal axis of the papilla is oblique. The explanation of this is that in the majority of cases we do not look directly at the papilla, in consequence of the ectasia of the posterior pole, but at it in half profile, as has been shown by *Dimmer*. The papilla lies to the nasal side of the posterior pole of the eye; the more this is stretched the more the papilla moves toward the inner side of the cavity formed by the ectasia; if the ectasia is strictly on the temporal side of the papilla, the latter lies strictly on the nasal side of the former, and the foreshortening then affects only the horizontal axis (Fig. J), but if the ectasia is downward and outward, the papilla clings to the upper inner wall of its cavity, and the foreshortening takes place in an oblique axis (Fig. K). As the atrophy of the chorioid occurs chiefly in the places where the stretching is greatest, this relation between the form of the staphyloma and that of the papilla is readily understood. If the ectasia is situated on the nasal side of the posterior pole the papilla occupies the floor of the hollow and consequently we look directly at it, and as the hollow made by the ectasia is on all sides of the papilla, the staphyloma is circular (see Fig. L).

### 3. Conus inferior (Fig. 8).

This is a special form of conus which is to be regarded as a rudimentary coloboma of the chorioid. Like the latter, it lies below the nerve, and is usually associated with a change in the form of the papilla, which is, in most cases, obliquely or transversely oval, and seems to be smaller than normal. The dividing line between the conus inferior and the papilla frequently is not as distinct as it is in Fig. 8. Anomalies in the nature of the excavation and in the subdivision of the vessels are often present. In almost all cases, though to a greater degree in some than in others, the color of the fundus is uniform in its upper part and almost albinotic in its lower. Astigmatism and amblyopia are usually associated with this form of conus.

The conus inferior differs plainly from the staphyloma posticum and the conus myopicus not only in its position, but also in the absence of scleroses in the vessels of the chorioid and in the form of the papilla.

### 4. Peripapillary Atrophy of the Chorioid (Fig. 65).

Peri- and parapapillary atrophy of the chorioid, the result of arterio-

sclerosis, is quite similar in appearance to staphyloma posticum. It is characterized by white cords formed by the sclerotic vessels of the chorioid, while the sharply outlined form of a crescent is generally absent. Its margins are indistinct, and it sends out narrow projections, like feelers, into its neighborhood, as shown in Fig. **65**. It is only in exceptional cases that this sclerosis takes the form of a staphyloma (Fig. **7**).

5. The **Halo** (Fig. **14**).

The halo is a more or less broad, complete or partial, yellowish-gray circle which is immediately adjacent to the papilla, and fades away into its surroundings with an indistinct margin. It is met with in old people, the halo senilis, and in glaucoma, the halo glaucomatosus. The halo corresponds to an atrophy of the chorioid and of the pigment epithelium, yet no sclerotic vessels can usually be seen within it ophthalmoscopically. Sometimes the ring has a more yellowish, or reddish tinge of color, which may be caused by the simultaneous presence of an exudate between the chorioid and the retina.

The halo glaucomatosus can readily be diagnosed as such when the course of the retinal vessels is noticed. A single vessel that bends sharply over the margin of the papilla suffices to make the diagnosis of glaucoma positive and to reveal the true nature of the ring. On the other hand, the halo senilis can be readily distinguished from staphyloma by the absence of visible vessels of the chorioid, from the scleral ring by the indistinctness of its margins, and from the tissue of the papilla by its color.

6. **Medullated Nerve Fibers** (Fig. **9**).

The fibers of the optic nerve are accustomed to lose their medullary sheaths before they pass through the lamina cribrosa and consequently become transparent, but in exceptional cases the fibers retain their sheaths as they spread out in the retina and hide all the tissues beneath them with a mantle that is sometimes yellowish white, sometimes bluish white.

The color varies according to the density and number of the fibers. It is a pure white when the fibers of the entire layer are opaque; it is a reddish yellow when the density of the fibers is slight, in consequence of the color of the subjacent portion of the fundus.

It happens very rarely that the medullary sheaths of the *orbital* segment of the optic nerve continue without interruption into the intraocular portion; in most of these cases the sheaths discontinue at the lamina cribrosa and reappear a little farther on, as the fibers spread out in the papilla or the retina.

These fibers usually adhere to the papilla and lie above or below it for the most part, very often covering the corresponding margins. The uncovered portion of the papilla then appears particularly dark red from the effect of contrast. These fibers never have a sharp margin, but send flamelike processes into the normal retina. Usually a distinct, radiating striation can be

perceived in the white area. The deep vessels of the retina may be completely covered by these medullated nerve fibers, while the superficial ones may jut out more or less sharply from the white mass (see page 102).

This behavior of the vessels of the retina, the indistinct border, the color and the striation are sufficient to differentiate medullated nerve fibers from conus, staphyloma, and peripapillary atrophy; the absence of all other pathological changes, such as hemorrhage and œdema, as well as its immediate connection with the papilla, serve to distinguish them from exudates and from patches of degeneration (see page 128).

Rabbits (Fig. **86**) always have medullated nerve fibers in their retinæ, horses have them very often.

# PLATE III

Fig. 4. Conus Temporalis

Fig. 5. Conus Temporalis, Supertraction in "School" Myopia

## Fig. 4.   Conus Temporalis

(See page 33)

Next the temporal margin of the disc of the optic nerve is a narrow white crescent which is separated from the rest of the fundus by a sharply defined edge. The papilla plus the crescent is surrounded by a strongly pigmented ring. For the anatomical explanation of such a crescent, see page 34. An artery arises from the line of delimitation between the papilla and the conus, which does not come from the central artery of the retina, but from *Zinn's* arterial plexus, and is a cilioretinal artery (see page 23).

The head of the optic nerve shows a distinct excavation in its temporal portion.

## Fig. 5.   Conus Temporalis, Supertraction in "School" Myopia

(See page 34)

At the temporal margin of the head of the optic nerve is a crescent some-what different in form and color from that shown in Fig. **4.** The excavation is very deep and the meshes of the lamina cribrosa are to be seen distinctly in its floor. The sclera is drawn over the nasal margin of the papilla, in consequence of which its outline is indistinct (see page 34). The fundus is uniformly pigmented, on the whole, but at certain places the vessels of the chorioid may be seen through the pigment layer.

Tab. **3.**
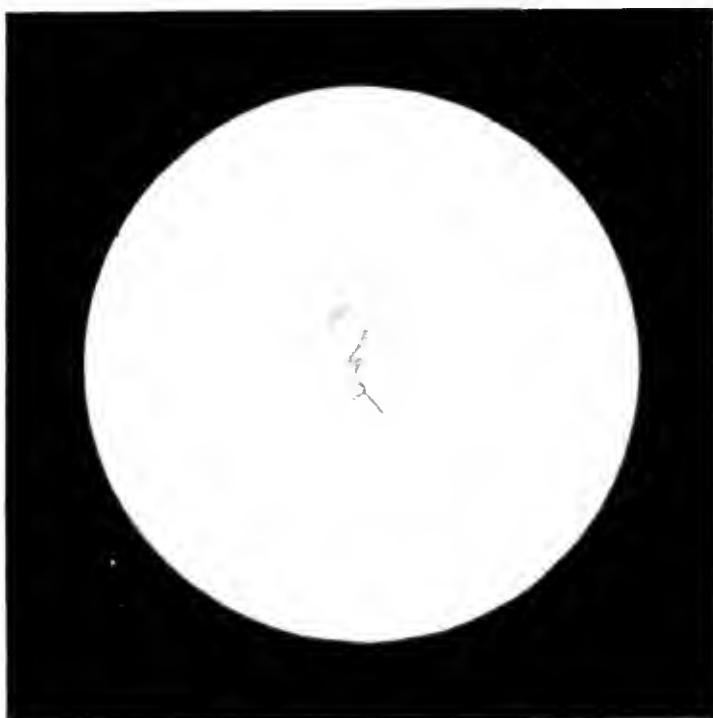


Fig. 4.
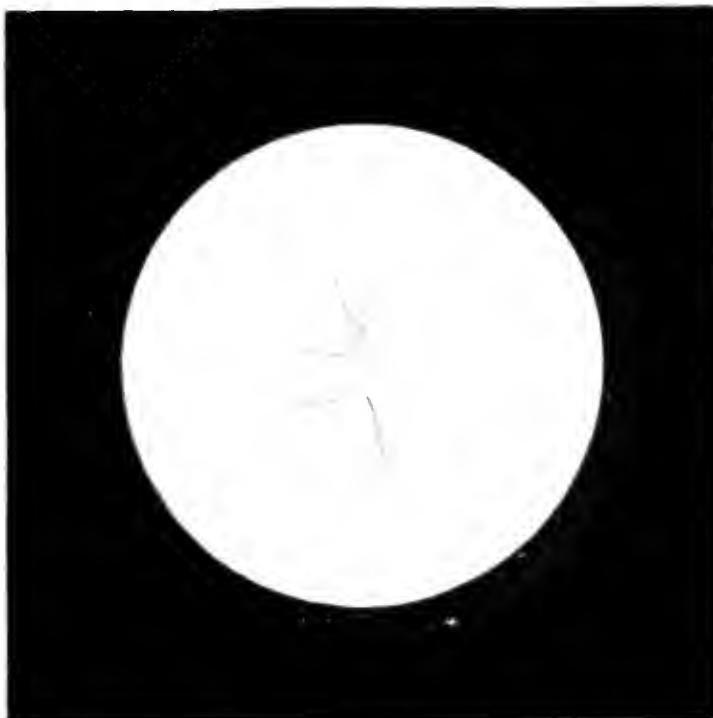


Fig. 5.

# PLATE IV

Fig. 6.  Commencing Staphyloma Posticum in "Congenital"
Myopia

Fig. 7.  Crescentic Sclerosis of the Chorioid Due to
Arteriosclerosis

## Fig. 6. Commencing Staphyloma Posticum in "Congenital" Myopia

(See page 37)

The term staphyloma posticum is made to include not only the outward bulging of the eyeball at its posterior pole, but also the change in the chorioid induced by it close to the head of the optic nerve. The epithelium of the retina, which is not very thick elsewhere in the fundus (tesselated fundus, see Fig. **2**), has undergone total atrophy over the area of a parapapillary crescent, and so allows the markings of the chorioid to be seen distinctly. The visible vessels of the chorioid are almost wholly obliterated, so that the chorioidal pigment which lies between them comes out very plainly. If this pigment also should disappear in the course of the disease the crescent would become pure white and resemble the conus in Fig. **4** (compare with the schematic drawing in Fig. **54**). The neighboring vessels of the chorioid appear to be diseased, an indication that "congenital" myopia has a progressive character. This picture was taken from the eye of a boy 10 years old, who had 12 D of myopia (see page 37).

The papilla itself and the retinal vessels are normal.

## Fig. 7. Crescentic Sclerosis of the Chorioid Due to Arteriosclerosis

(See page 39)

A picture can be produced by arteriosclerosis that closely resembles the preceding. The presence of pigment between the sclerotic vessels of the chorioid is only suggested. This picture was taken from the eye of a man 68 years old, whose refraction was emmetropic. As the sclerosis of the vessels advances such pictures may be created as those shown in Fig. **65**.

Tab. 4.
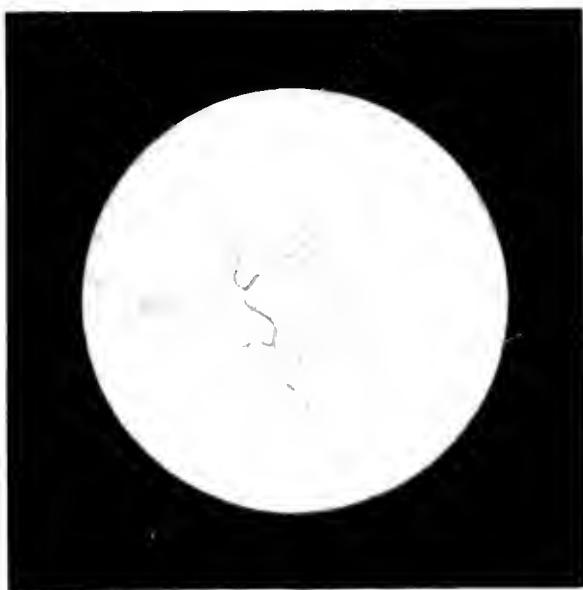


Fig. 6.
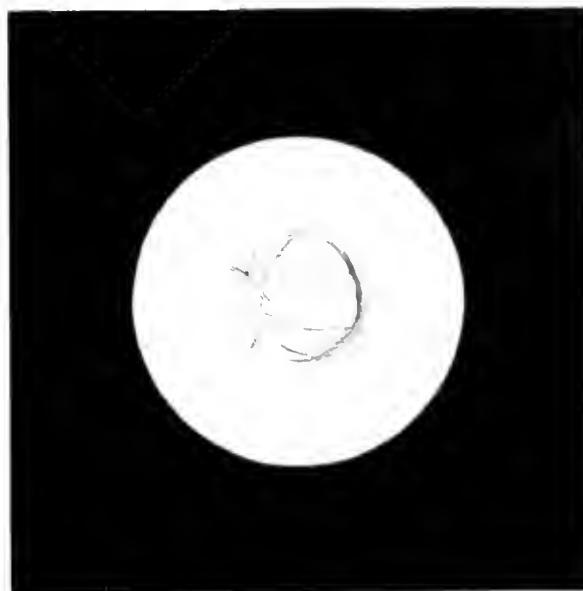


Fig. 7.

## PLATE V

Fig. 8.   Conus Inferior; Partial Albinism

Fig. 9.   Medullated Nerve Fibers

## Fig. 8.  Conus Inferior; Partial Albinism

(See page 39)

The papilla has a remarkably transversely oval form which is completed into a circle by the adjacent, or rather subjacent, crescent.

The line of demarcation between the papilla and the conus is not always as sharply defined as it is in this case. Dark spots can be seen scattered about in the conus (see page 39).

The portion of the fundus in the vicinity of the conus is distinctly albinotic, while the rest of it is uniformly colored.


## Fig. 9.  Medullated Nerve Fibers

(See pages 40 and 128)

Medullated nerve fibers ordinarily lie, as in this case, in immediate connection with the papilla and extend out from it. The papilla seems to be redder than usual from the effect of contrast. The fibers conceal whatever lies beneath them, e.g., the more intense color of the pigment epithelium generally to be found in the vicinity of the papilla. As they overlie in part the vessels of the retina the latter appear to be narrowed in certain places. They form a mass that is striated, is brilliant white, and that often ends in delicate, separate fibers which look like white hairs. In rare cases these patches may be of considerable extent, but, almost invariably they follow the courses of the vessels of the retina. Although they are usually in immediate connection with the papilla, they may be found separate from it in exceptional cases. For the *differential* diagnosis from other conditions, particularly from albuminuric retinitis, see page 128. These fibers form an absolutely harmless congenital anomaly. The rest of the fundus is normal.

Tab. 5.
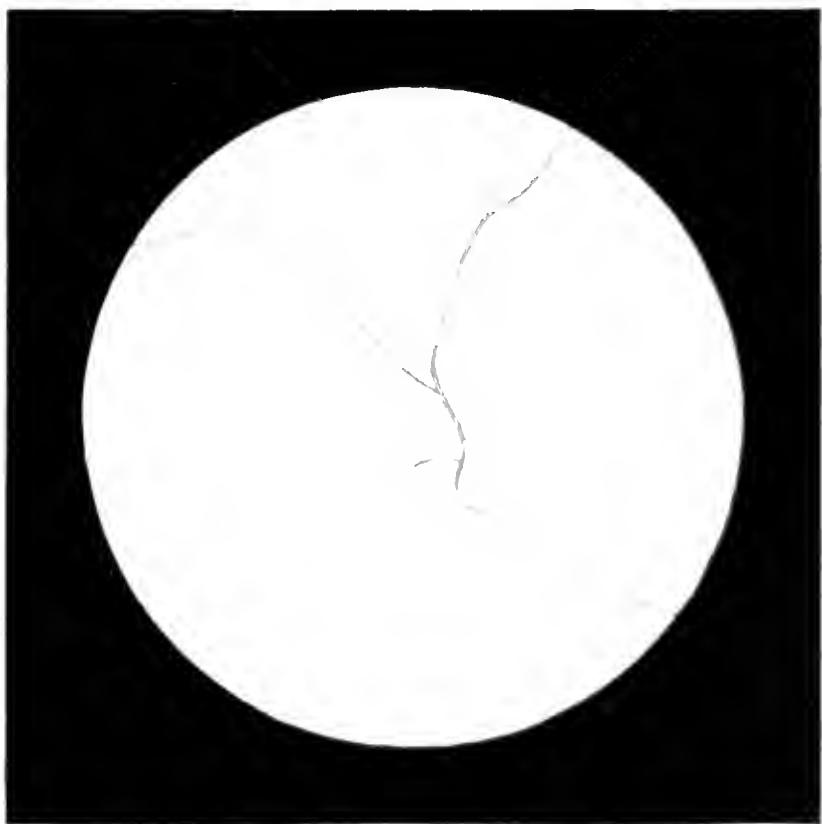


Fig. 8.
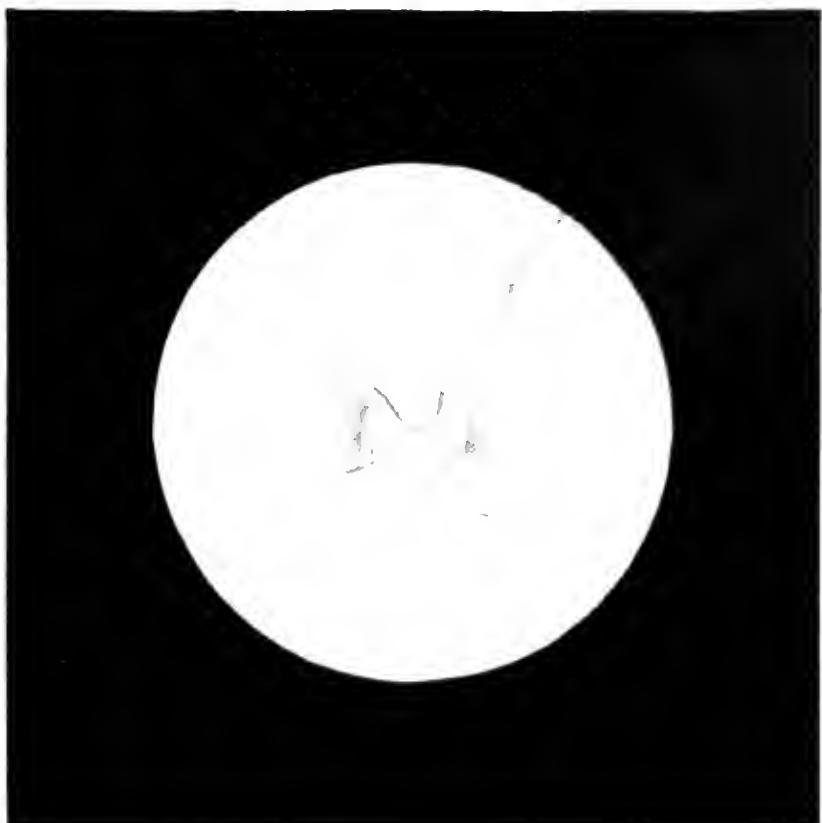


Fig. 9.

# Atrophy

## Atrophy of the Optic Nerve

# ATROPHY

The changes in the optic nerve are to be considered mainly from 4 points of view for the purpose of diagnosis:

1. **Color and Transparency.**
2. **Margins.**
3. **Conditions of Level.**
4. **Conditions of the Vessels.**

The alterations in color and in the margins are apt to be the most significant, so the changes in the optic nerve will be grouped first from these points of view.

## Whiteness of the Optic Nerve.

It must be understood from the start that the ophthalmoscopic condition alone does not suffice to establish the diagnosis in many cases, but that the simultaneous determination of the vision and of the visual field is imperatively demanded.

The use of indirect illumination is often of value in cases of commencing atrophy. For this purpose the mirror is turned a little to one side, while the observer is looking at the papilla, so that the apex of the cone of light falls only on the margin of the papilla. Slight changes in the color of the latter then become plainly visible.

A guide to the differential diagnosis of the various forms of atrophy can be found on the next page. It may, perhaps, appear too schematic to many; I readily admit that in individual cases the differential diagnosis, for example, between nutritional and simple atrophy, is by no means as easy as might be supposed from the chart, but yet the medium types can be distinguished by it, and this forms its justification.

Some remarks concerning the so-called *atrophic excavation* may be in order before passing to the consideration of the individual forms of atrophy.

Following the lead of *v. Jaeger*, many authors speak of an atrophic excavation, yet *Elschnig* is right when he maintains that no such picture can be seen either with the ophthalmoscope or with the microscope, and that its occurrence cannot be understood pathologically, because the nerve fibers alone disappear in atrophy, leaving the stroma and connective tissue, the latter of which is sometimes increased. When a large physiological excavation

## DIFFERENTIAL DIAGNOSIS OF THE VARIOUS FORMS OF ATROPHY

| | Color | Margin | Conditions of Level | Vessels | Visual Field |
|---|---|---|---|---|---|
| Tabetic atrophy | gray white white | distinct | normal | normal | loss of sectors, concentric contraction |
| Atrophy from interruption of conduction | white | distinct | normal | normal | usually amaurosis is present |
| Nutritional atrophy (atrophy after occlusion of arteries) | white | distinct | normal | arteries small, threadlike when occluded | variable (amaurosis after occlusions) |
| Glaucomatous atrophy | gray white | distinct, sometimes with a halo | deep, abrupt excavation to the margins of the nerve | vessels bent like hooks at the margin of the papilla; veins engorged and tortuous; arterial pulse | nasal contraction; more rarely concentric contraction |
| Neuritic atrophy (atrophy after choked disk) | white | indistinct | normal; after choked disk rather elevated papilla | veins engorged, tortuous, and sheathed | variable |
| Retinitic atrophy | pale yellow, or dirty reddish to gray yellow | obscured | normal | threadlike | very great concentric contraction |
| Atrophy of the papillomacular bundle from toxic causes and multiple sclerosis | temporal side of papilla white or gray white, the rest normal | distinct | normal | normal | central scotoma for green and red, later for all colors |

(The first two rows are bracketed as **Simple atrophy**.)

already exists it may seem to become enlarged by the rounding off of its margins, especially when the difference of color between the papilla and the floor of the excavation disappears as the atrophy progresses; but even in such a case it is better to speak of an atrophy with a large physiological excavation, than of an atrophic excavation.

We have to distinguish between **total and partial atrophies.** This can apply naturally only to the completed condition characteristic of the clinical picture, for in commencing total atrophy the process will be most marked at the place where the nerve fibers are weakest, where a brighter tone of color prevails, i.e., on the temporal side of the papilla, so that in this stage the total may present the appearance of the partial atrophy. On the other hand, a partial atrophy that has existed for years may become total.

*It follows from what has been said, and from what is yet to come, that we must not be contented with the diagnosis "atrophy," but must try to ascertain the specific diagnosis of simple, neuritic, and other forms, from the symp-*

*toms that are present. This will not prove difficult to those who are accustomed to consider the papilla always from the 4 points of view, to take into account its color, margins, level, and vessels.*

## A. Total Atrophy

1. *Simple Atrophy* [1] (Figs. **10** and **11**).

Simple atrophy presents the following characteristics:

Color: white to gray white.

Margins: [2] normal.

Level: normal.

Vessels: normal.

Hence, the color alone is changed.

The retinal vessels may become smaller in the later stages, so as to resemble nutritional atrophy, but this does not belong to the typical picture.

What etiological conclusions can be drawn from such a condition? Unfortunately, very many. The atrophy may be of a true neurogenous origin, caused by cerebral disease, when it is primary, or it may be due to injuries or compressions, when it is secondary. We see the same picture in both cases. The true diagnosis will depend therefore on the findings in the general and neurological examination, unless we are able to find other points in the eye that are of diagnostic assistance. Primary atrophy is the same as that which has frequently been termed gray atrophy, but—we may almost say again unfortunately—the color has no signification in the differential diagnosis. In the first place, the gray tone of color is met with comparatively seldom; in the second, it has been seen many times in other forms; in the third, it may disappear and give place to a white tone after it has once been observed, or the reverse may take place. As the other ophthalmoscopic details, the margins, level and vessels show no deviation from the normal in typical cases, we have to include under the caption of simple atrophy clinical pictures which are etiologically very different. This is why it is so important to notice all other ocular symptoms, and to investigate the general condition.

**Etiologically,** tabes is the first disease to be thought of; then come general paralysis and syphilis. Syphilis of the optic nerve usually appears in the form of a neuritis, or of a neuritic atrophy, yet syphilitic diseases of parts that are situated more centrally, such as meningitis, gumma of the chiasm, and hydrocephalus, may cause a simple atrophy secondarily. Tabes surpasses everything else in importance. Tabetic atrophy begins, as a rule, very early in the disease and may be for years its only symptom. It rarely

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[1] A number of animals have perfectly white papillæ, for example, the rhinoceros, the armadillo, the porcupine, the anteater, and the hedgehog.

[2] When a conus or a staphyloma is annexed to the atrophic papilla it may be differentiated from the latter by the fact that it presents a peculiar, rather yellowish white, instead of the white of the papilla.

appears at the same time with the ataxia of the lower limbs. Out of the large number of patients that I have seen who were blinded by this disease, I can recollect only a very few who had a high degree of ataxia. Although it is to be feared that a tabetic who closes his eyes will lose his balance and fall, because of his ataxia, these blind persons, in whom likewise the sense of sight for orientation is absent, go about quite well, and present the same appearance as other blind persons. Tabetic atrophy almost always affects both eyes and leads to blindness, though with remissions.

*Tabes frequently causes an ocular triad; atrophy of the optic nerve, paresis of the ocular muscles, reflex immobility of the pupils.*

**Differential diagnosis.** When we find a reflex immobility of the pupil associated with a simple atrophy of the optic nerve, the case is one of tabes; when the immobility of the pupil is absolute, i.e., when the pupil does not react to either light or convergence, especially when the pupil is also dilated, general paralysis or syphilis is probably present. Meiosis occurs only in tabes. On the other hand, the reactions of the pupil may be preserved in syphilis, while this is almost never the case in tabes. A paresis of the abducens, or a paresis of a portion of the oculomotorius, is indicative of tabes, while a facial paresis, especially in connection with symptoms of hemiplegia, points rather toward general paralysis.

Sometimes syphilis appears quite like tabes in its accompanying symptoms, and then the differential diagnosis may be very difficult, the more so as *Wassermann's* reaction is often positive in tabes. Atrophy of only one optic nerve, an accompanying hemianopsia, *total* internal ophthalmoplegia, *bilateral* paresis of the oculomotor nerve, or bilateral ptosis, generally indicate that the disease is syphilitic rather than tabetic.

The field of vision in tabes usually shows sectorlike, or concentric losses. A central scotoma with total atrophy is indicative of syphilis, with partial atrophy of multiple sclerosis.[1]

None of the other causes are very frequent. Among them may be mentioned as particularly important, interruption of the conductivity of the optic nerve (Fig. **10**) by direct or indirect injuries, as in fracture of the base of the skull, compression in the optic canal, as in oxycephalus, slowly growing tumors at the base of the skull, hydrops of the third ventricle, and pressure of the arteriosclerotic carotid upon the intracranial portion of the nerve.

**Pathologically,** simple atrophy of the fibers of the optic nerve takes place without material proliferation of connective tissue; both the vessels

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[1] In the rare cases of tabes with central scotoma there is a concentric contraction at the same time; this is not present in multiple sclerosis. Another difference between tabes with central scotoma and multiple sclerosis is that in the former the papillæ are pale at a time when the vision is still good, while in multiple sclerosis it is the vision that is lost first, it may be rather suddenly, and the papilla subsequently becomes pale.

and the capillaries are preserved. Hence the white or gray discoloration of the papilla cannot be caused in this way. It is probably due to the changed optical conditions induced by the atrophy of the nerve fibers, which make it possible for the lamina cribrosa to reflect the light that falls upon it more sharply and in greater quantity than it can under normal conditions.

### 2. *Nutritional Atrophy.*

The appearance of this may be extremely like that of the atrophy just described. Only the absolutely negative evidence of a neurological and internal examination, with the exception of an arteriosclerosis that is often moderate, together with the condition of the retinal vessels, proves the diagnosis. As is to be expected in arteriosclerosis, this form of atrophy is usually met with in old people. It is not rarely found in company with arteriosclerotic vessels in the chorioid, which often surround it like a staphyloma (see page 39). For this reason the margins of the papilla appear somewhat indistinct in many cases, but this is due only to the absence of contrast. Otherwise the margins in this form are sharply defined, the level is normal, the vessels alone are altered. In many of these cases the first things that can be seen by accurate observation are irregularities of caliber, thickenings of the walls, and obliterations of the smaller vessels, such as will be described later under "The Changes in the Vessels of the Retina," and are considered to be characteristic of arteriosclerosis of these vessels. The visual disturbances are comparatively trivial in these cases, and the atrophy caused by chronic disturbances of nutrition are differentiated by this fact from the

## Atrophy Due to Occlusion of the Arteries

The picture of acute occlusion of an artery is shown in Figs. **44** and **45,** and is described on page 150. After the acute symptoms have passed away a condition develops which is illustrated by Fig. **16.**

The arteries are threadlike, no longer visible in some places, the optic disk is white, with normal margins and level; usually, though not always, small patches of degeneration are found in the macula, which are called coronulæ and are described on page 127.

The vision is usually totally lost in these cases.

### 3. *Glaucomatous Atrophy* (Fig. **14**).

The color in this form of atrophy is a gray white rather than a pure white, as a rule; the margins of the papilla are sharply defined, but in many cases they seem to be obscured by a surrounding ring, the so-called halo. We should observe carefully where the papilla stops and the halo begins, the line of separation can usually be recognized plainly from the difference in color between the two. The vessels are rarely quite normal, the veins are usually broad, sometimes varicose, while the arteries are engorged only at first, frequently pulsate, but later appear to be contracted.

This form is sharply differentiated from all others by the behavior of the vessels at the margin of the papilla. As can be plainly seen in Fig. **14,** the course of the vessels up to the margin of the optic disk is perfectly



Fig. M. Glaucomatous Excavation.

The excavation begins sharply at the margin of the papilla. The optic nerve is laterally pouched out on its nasal side. When we look from in front upon a vessel that dips down at this place it will not be visible as it courses along this pouch, but will apparently come to an end at the margin of the papilla. The optic nerve itself is reduced by atrophy. These pathological conditions correspond to what is seen ophthalmoscopically in Fig. **14.**



Fig. N.—Deep Physiological Excavation.

In contrast to the preceding drawing the excavation does not begin at the margin of the papilla, but normal tissue lies between the margins of the disk and of the excavation. A vessel coursing over the margin of the excavation may bend in a manner similar to that followed by those in a glaucomatous excavation, but this bend does not take place at the margin of the papilla. The corresponding ophthalmoscopic picture is Fig. **15.**

normal, but there they bend suddenly and seem to disappear in the cavity; it is only by a change of focus—in the direct method by the interposition of a concave glass—that they can be seen in the floor of the excavation. From the difference in focus and from the parallactic displacement (see page 9) between the vessels on the margin of the papilla and those on its floor, the difference of level between the excavated papilla and its surroundings can be determined. The floor of the papilla usually lies 1 mm behind the level of the retina. The fact that it is abrupt and extends to the margin distinguishes the glaucomatous from the physiological excavation. Whenever a vessel is seen to make a distinct hook over the margin of the papilla the diagnosis of glaucoma is justified. Usually the excavation is total. The markings of the lamina cribrosa are very plainly visible in fresh cases, but in older ones they are hidden by the proliferation of connective tissue. It may be said here that the color of the optic nerve need not be abnormal in the commencement of a glaucoma, but that the excavation and the behavior of the vessels suffice for the diagnosis. Moreover, under the influence of an antiglaucomatous treatment the excavation undergoes involution in many cases.

In old glaucomatous eyes we find obliterations of the vessels throughout entire areas of the retina, as well as a new formation of large vessels at the margin of the papilla, or in the excavation; sometimes junctions between the retinal and chorioidal vessels, the so-called opticociliary vessels, are also to be seen (see page 104).

4. *Neuritic Atrophy* (Figs. **12** and **13**).

Neuritic atrophy is clearly distinguishable from all of the other forms by the indistinctness of the margins of the papilla, and by the invisibility of the apertures in the lamina cribrosa.

In the atrophy that follows neuritis, or choked disk, the papilla is at first white gray with striated or obscure margins, but this color changes pretty soon into a pure white. The margins remain indistinct and striated; no excavation and none of the details of the lamina cribrosa are to be seen. The surface of the papilla usually rises a little above the level of its surroundings, to a fairly high degree when the atrophy is due to choked disk.

The vessels are always changed; the veins are tortuous, engorged to a greater or less degree, while the arteries are smaller than normal. This difference is particularly marked in old choked disk (see Fig. **13**). In the majority of cases the vessels are also accompanied by white stripes.

Newly formed vessels, which are frequently twisted like corkscrews, or formed into loops, are not rarely seen on the papilla; opticociliary vessels (see page 104) are also observed more often than usual.

These phenomena become the more marked the longer the inflammation has lasted. In cases that run a rapid course a neuritic may therefore sometimes appear very like a simple atrophy, and inflammations that occur in

old age are apt to have severer sequelæ than those that affect younger persons.

The differences between the atrophies caused by inflammation and by choked disk equalize themselves in the course of time, especially in young people, so that it is sometimes impossible to make a differential diagnosis between these two forms.

If the inflammation was not confined to the papilla, but involved its surroundings, discolorations and partial vascular changes of particularly high degree are accustomed to appear as sequelæ. On the other hand, there is in many cases an entrance of pigment into the atrophic retina, as shown in Fig. **12**, which causes an uncertain grayness about the optic nerve.

*Pathologically,* the indistinctness of the margins of the papilla, as well as the stripes that accompany the vessels, are to be ascribed to a proliferation of the glia tissue, though the stripes along the vessels may be caused in part by changes in the tissue of the walls of the latter.

*Etiologically,* all of those factors have to be taken into account which can cause an optic neuritis.

The so-called retrobulbar neuritis can never cause a neuritic atrophy.

5. *The Atrophy of the Papilla in Retinitis Pigmentosa* (Fig. **51**) is to be considered only as one symptom of a clinical picture, but, as it is usual for the glance of the observer to fall first on the papilla when he is making an ophthalmoscopic examination, its characteristics may be mentioned here.

Retinitic atrophy has a certain resemblance to the neuritic form in that the margins of the papilla are indistinct; its color, however, is commonly a yellowish gray, rather than a pure white.

What is particularly marked is the great diminution of the size of the retinal vessels, which ordinarily show no changes in their walls; in this respect it may resemble nutritional atrophy, especially when due to occlusion of the central artery, but it may be differentiated from this by the condition of the margins, which usually are sharply defined in nutritional atrophy, indistinct in retinitic, and by the color, which is white in the former, yellowish in the latter.

The condition of the retina, especially in the periphery (see page 174), is decisive as regards the diagnosis.

## B. Partial, or Temporal, Atrophy of the Optic Nerve

The margins, level, and vessels are perfectly normal; the only variation from the normal to be seen is the paleness of the temporal side of the papilla. As large physiological excavations situated in this portion may simulate a temporal paleness under certain circumstances, and, on the other hand, as the temporal portion of the disk is normally much brighter than the nasal, it is evident that great care must be exercised in making the diagnosis.

If the diagnosis cannot be made positively from the ophthalmoscopic

picture, the field of vision is to be investigated. If a central scotoma [1] is found, with the outer portions of the field normal, which is at first only for green and red, later for white also, a positive diagnosis of partial atrophy may be made.

The determination of the **etiology** is of very great importance, as the temporal paleness may be caused by very different diseases.

1. *Chronic intoxications.* Preeminent among these is poisoning with alcohol and tobacco. It is still a question whether tobacco alone can produce such an effect. Then follow the toxic effects of methyl alcohol, lead, bisulphide of carbon, atoxyl, quinine, filix mas, and arsenic, as well as of auto-intoxications, particularly in diabetes.

2. *Multiple sclerosis.* The visual disturbance caused by this condition is present in about half of the cases of multiple sclerosis, and may precede by years all other signs. Nystagmus when the eyes are turned as far as possible to one side or the other is frequently present as an accompanying symptom, paresis of the ocular muscles are less common, and an immobility of the pupil is almost never seen (see page 54).

3. The *inflammatory diseases of the posterior ethmoidal cells and of the sphenoidal sinus* may bring about a similar picture through an extension of the inflammation to the optic nerve, or through the influence of toxines some time after its subsidence.

It follows from the nature of the cause that the disease regularly affects both eyes in Case 1, while in Cases 2 and 3 only one eye may be affected.

**Pathologically** this is to be considered as a secondary atrophy of the papillomacular bundle of optic nerve fibers, which passes over the temporal margin of the papilla and supplies the macula; hence the central scotoma. The disease actually begins in the ganglion cells of the retina.

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[1] The stereoscopic method of *Haitz* for the determination of a central scotoma is very simple and valuable (see page 79).

# Fig. 10.  Simple White Atrophy of the Optic Nerve
(See page 53)

When we study systematically the color, margins, level and vessels of the papilla we see that its color is white, except for some stippling which indicates the apertures in the lamina cribrosa, that its margins are distinct, that its level is the same as that of the retina, and that its vessels are normal. Therefore everything about it is normal except its color, so that, by applying our schedule, we are led to the diagnosis of simple atrophy. Hence we have to think first of tabes, but in this case the result of the neurological examination was negative. The history stated that a stick had penetrated the orbit of the patient at a time when he was stooping, and that the eye had been made blind immediately. The diagnosis therefore was atrophy after interruption of conductivity (see page 54).

The fundus is of the tesselated type, the retina is more strongly pigmented in the region of the macula, distinct reflex striæ are visible along the vessels.

# Fig. 11.  Simple Gray Atrophy of the Optic Nerve
(See page 53)

As in the preceding picture the color of the disk is the only deviation from normal; the margins, vessels and level are normal. The dark spots in the fundus are caused by the pigment in the chorioid showing through the retina (tesselated fundus). This patient had tabes. In the later course of the disease the gray became brighter, and the vessels smaller.

Tab. 6
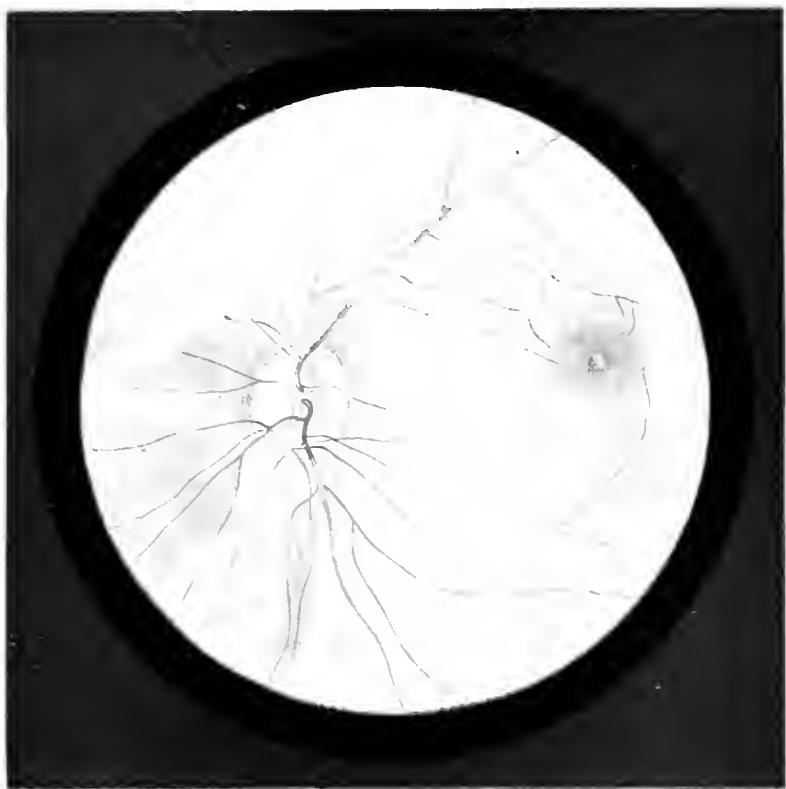


Fig. 10.



Fig. 11.

PLATE VII

Fig. 12. Atrophy after Inflammation of the Optic Nerve,
Neuritic Atrophy

Fig. 13. Atrophy of the Optic Nerve after Choked Disk

## Fig. 12.   Atrophy after Inflammation of the Optic Nerve, Neuritic Atrophy

(See page 57)

The papilla is white, its margins indistinct, the excavation and the markings of the lamina cribrosa are erased, the vessels are bordered by white stripes.

The indistinctness of the papilla is due to a proliferation of glia which does not cease exactly at its margin and fills the cavity of the excavation. The stripes along the vessels are caused partly by a proliferation of glia, partly by changes in their walls (see page 58).

Pigment has migrated into the retina, which has been rendered atrophic by the inflammation, and has produced the gray halo about the papilla.

The cause of the optic neuritis in this case was syphilis, which had been acquired 3 years before.


## Fig. 13.   Atrophy of the Optic Nerve after Choked Disk

(See page 57)

As in the preceding case, the margins of the papilla are indistinct; it can be seen from the ring surrounding the disk how far the swelling extends into the retina.

The papilla is distinctly elevated, as is shown by the mounting of the vessels at its margin; the vessels themselves show the disproportion in size between the arteries and veins characteristic of choked disk.

The vision was much impaired in this case.  The other eye of the patient, who died of gliosarcoma of the cerebellum soon after the completion of the picture is shown in Fig. **25.**

Tab. **7.**



Fig. 12.
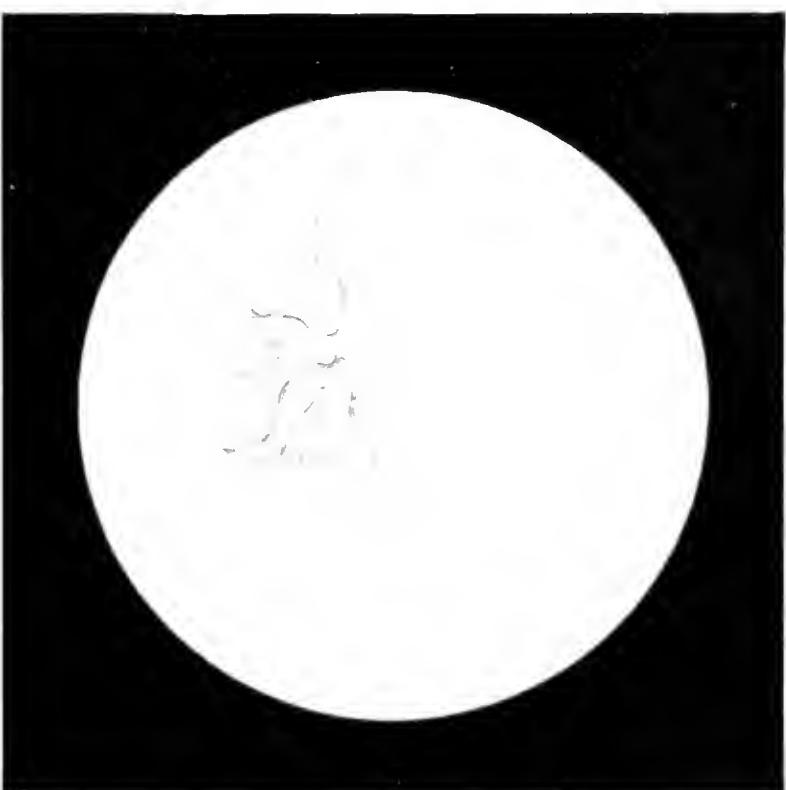


Fig. 13.

## PLATE VIII

Fig. 14.   Glaucomatous Excavation and Atrophy

Fig. 15.   Large Physiological Excavation

## Fig. 14. Glaucomatous Excavation and Atrophy

(See page 55)

The color of the papilla is gray in the center, with a gray green shadow tone in the marginal portions, especially on the nasal side, which varies somewhat with the way in which the mirror is held, and is caused by the overhanging of the margins. The papilla is encircled by a sharply defined scleral ring, to which is added a yellowish gray ring which blends with its surroundings in a less distinct margin (halo glaucomatosus, see page 40). Its level is much deeper than that of the retina. The retinal vessels are not materially altered, but they break off and disappear exactly at the margin of the disk, and from this we conclude that the excavation is abrupt and extends to the margin of the nerve.

Compare with this the pathological drawing, Fig. M in the text.

The rest of the fundus is uniform and stippled. The vision in this case had fallen to one sixth of the normal, and there was a considerable concentric contraction of the visual field, particularly marked on the nasal side.

## Fig. 15. Large Physiological Excavation

(See page 56)

In contrast with the preceding picture we see here a narrow zone of normal tissue between the margins of the papilla and of the excavation.

The color, margins and vessels of the papilla are normal.

The vessels of the retina do not stop at the margin of the excavation, but can be plainly traced, although their direction is changed. We conclude from this fact that the sides of the excavation are not precipitous, as in the last picture, but that they slope gradually, like the sides of a cup.

The holes in the lamina cribrosa can be seen very distinctly in the floor of the excavation.

Compare with this the pathological drawing, Fig. N, page 56.

66

Tab. 8.
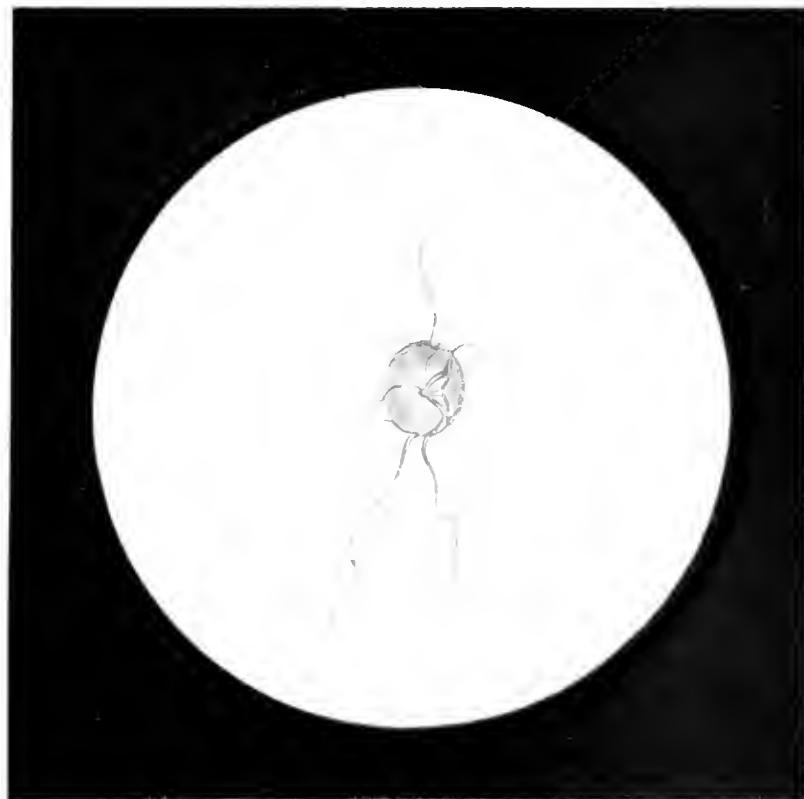


Fig. 14.



Fig. 15.

## PLATE IX

Fig. 16.  Atrophy of the Optic Nerve after Occlusion of the
Central Artery

Fig. 17.  Partial, or Temporal, Paleness of the Optic Nerve

# Fig. 16.  Atrophy of the Optic Nerve after Occlusion of the Central Artery

(See page 55)

The papilla is white, its margins are fairly distinct, its level is the same as that of the retina, its veins are of approximately normal size, its arteries are threadlike.

In the vicinity of the optic nerve is to be seen again an uneven coloring of the fundus as the consequence of an immigration of pigment into the atrophic retina.

In the macula is to be seen, framed in pigment, a coronula of little bright points, which is characteristic of an occlusion of the artery that took place at some previous time (see page 127).

The vision of this eye was totally lost.

As a general rule the arteries are completely empty of blood for a few days after the occlusion, and then gradually refill from the ciliary vessels by way of *Zinn's* arterial plexus (see page 168).

# Fig. 17.  Partial, or Temporal, Paleness of the Optic Nerve

(See page 58)

The margins, vessels and level of the papilla are normal, but its temporal half exhibits an abnormal paleness that far surpasses the ordinary difference in color of the temporal from the nasal portion.

This patient had at the same time a central scotoma.  The neurological examination revealed that he was suffering from multiple sclerosis.  The loss of vision caused by the central scotoma was the only subjective symptom of which he complained.  The discovery of the temporal paleness led to the neurological examination that disclosed the presence of this serious disease.

Tab. **9.**
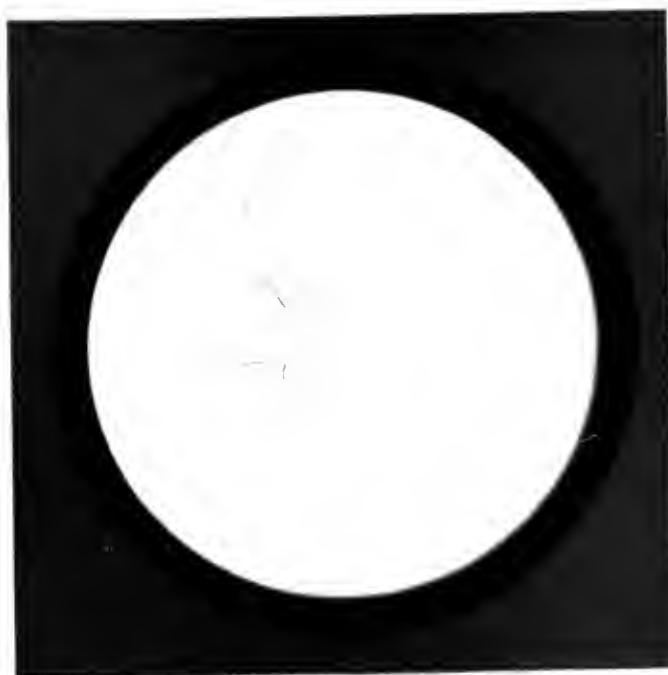


Fig. 16.



Fig. 17.

Abnormal Redness of the Papilla. Optic Neuritis, Retrobulbar Neuritis, and Choked Disk

# Abnormal Redness of the Papilla, Optic Neuritis, Retrobulbar Neuritis, and Choked Disk

## I. Redness of the Papilla by Itself

without any other symptom is a condition that must be considered with extreme care, just the same as paleness, because the color of the papilla varies *physiologically* within rather wide limits and, moreover, is dependent on a number of secondary factors, with which it is necessary to be acquainted in order to estimate correctly the influence they exert. A papilla always appears redder in a blonde than in a brunette fundus; it appears to be redder when the light used is saturated with red rays, as for example that from a kerosene lamp; it looks redder in young than in old persons. It must also be remembered that a hyperæmia of the papilla may be induced by a prolonged examination with the ophthalmoscope, or by severe accommodative efforts in hypermetropes and presbyopes.

Aside from these causes, which may be termed physiological, there is a hyperæmia which appears as an *accompanying symptom of morbid processes*, as in inflammations of the anterior and posterior segments of the eye, especially in iritis and iridocyclitis, in injuries of the eyeball, in empyemata of the accessory sinuses, and in such circulatory disturbances as are caused by heart disease, or by tumors of the mediastinum, although the optic nerve itself is not diseased. The bearing of all these possibilities must be recognized and correctly estimated before a hyperæmia can be decided to be the *forerunner* of an *optic neuritis*, or of a *choked disk*. Finally, a particular form that is extremely apt to give rise to mistakes, and consequently has been termed pseudoneuritis, demands a special consideration.

**Pseudoneuritis** may appear with an obscuration of the margins of the disk, with engorgement and tortuosity of the vessels, and even with a slight prominence of the papilla, and yet the condition is not pathological, but congenital.

Hypermetropia, or hypermetropic astigmatism, is commonly present in these cases, and, as these conditions of refraction sometimes impair the vision, another symptom of true optic neuritis may be added, viz., the impairment of the vision. But pseudoneuritis can be positively differentiated from optic neuritis by the absence of the œdema about the papilla, which is present in almost all cases of optic neuritis and is always absent in pseudoneuritis. The presence of hemorrhages, or of disturbances in the field of vision, renders the diagnosis of optic neuritis certain, but these symptoms are absent in many cases.

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## II. Optic Neuritis

The differentiation of pseudoneuritis from optic neuritis is of so much the greater importance because the latter is an indication of the presence of some other grave disease, usually of the general organism. **Optic neuritis is not a disease per se, but is to be looked upon as a symptom of another serious disease.**

*Hence it is our duty in every case of optic neuritis to ascertain the fundamental disease which has produced it, and, first of all, to examine the urine and to test for Wassermann's reaction.*

The case would be parallel if we were satisfied with the diagnosis cough, and did not seek out the cause of the cough, should we neglect to make a general examination of the organism when an optic neuritis is present.

The causes are manifold and may be either general or local.

The commonest cause of an optic neuritis is *syphilis*, the next in frequency is *albuminuria*. All other causes are of secondary importance. They are tuberculosis, diabetes, the various forms of basilar meningitis due to acquired or hereditary syphilis or tuberculosis, the acute infectious diseases, such as typhoid fever, malaria, pneumonia, small pox, diphtheria, scarlet fever, epidemic cerebrospinal meningitis, and myelitis.

Among the local causes may be named suppurative inflammation in the orbit, in the ear, or in the accessory sinuses (see page 78), abscesses in the brain, and anomalies in the form of the skull, such as oxycephalus.

It should be noted that the inflammation excited by general disease is commonly bilateral, while that due to local causes is apt to be confined to one side.

**Diagnosis.**—If we follow our plan and study each papilla with regard to its color, margins, level, and vessels, we find the principal symptoms of optic neuritis to be:

1. Redness and cloudiness of the papilla.
2. Obscurations of its margins, peripapillary œdema.
3. Little or no elevation.
4. Little change in the arteries, broadening and tortuosity of the veins, which sometimes are provided with accompanying streaks.

Note to 1. The *redness* is caused by a congestion of the smallest vessels and its signification has already been considered.

Note to 2. The *cloudiness of the tissue of the papilla*, and the *obscuration of its margins*, is brought about by a marked œdema which permeates both the head of the optic nerve and the adjacent retina.

The *peripapillary* œdema forms a reddish gray, or pure gray ring, 1 or 2 papillary diameters broad, which surrounds the entrance of the optic nerve and obscures the tissue that lies beneath and within it. It is to be seen most plainly in an albinotic, or a tesselated fundus, because it hides the markings

# DIFFERENTIAL DIAGNOSIS BETWEEN OPTIC NEURITIS, CHOKED DISK, AND PSEUDONEURITIS

| | Color | Margins | Level | Vessels | Peripapillary (Edema of the Retina | Hemorrhages in the Retina | Vision and Refraction | Field of Vision |
|---|---|---|---|---|---|---|---|---|
| Optic neuritis | abnormally red and cloudy | partially or wholly obscured | little or no elevation | arteries little changed; veins broad and tortuous | always present | rarely present | usually much impaired | a central scotoma often present |
| Choked disk | | | great elevation | arteries small, veins enlarged; great disproportion in size between the veins and the arteries | always present | usually present | normal for a long time, often with transient obscurations | variable; normal, concentric contraction, sectorlike scotomas, hemianopsia [1] |
| Pseudoneuritis | | | little or no elevation | arteries normal, veins often broad and tortuous | never present | never present | normal, or impaired in consequence of hypermetropia or hypermetropic astigmatism | normal |

[1] For albuminuric choked disk, see page 79.

Aside from the forms of disease which even the expert finds difficult to differentiate, the diagnostic difficulties that arise may be avoided easily by proper technique and attention.

1. **Inaccurate focussing upon the ophthalmoscopic picture.** No one makes the diagnosis of optic neuritis more often than a neophyte in ophthalmoscopy, who has not yet learned to accommodate correctly upon the picture of the fundus and mistakes the papilla, which seems to him indistinct because of his faulty accommodation, for one that is pathologically changed. Usually the examiner is easily able to change the indistinct picture into a distinct one by moving his head *backward* or *forward,* keeping his accommodation unchanged (see page 5).

2. **Delicate, diffuse opacities of the cornea and lens** may likewise cause confusion by making the papilla appear indistinct. This error is easily avoided by an examination of the eye by *oblique illumination, or by throwing light into the eye with a mirror,* which should be made in every case previous to an attempt to see the fundus.

The source of error is not so easily to be detected in

3. **Diffuse opacities of the vitreous.** Usually it is extremely difficult to see these, but we may be aided if we examine the periphery of the fundus, as we always should. The cloudiness and obscuration caused by the œdema in optic neuritis extends at most one or two papillary diameters into the retina, which then assumes its normal character. The periphery of the fundus is normal in optic neuritis, although it may be obscured by opacities in the vitreous. When the latter are present, together with an optic neuritis, we have to take very careful note of the vessels, of any hemorrhages that may be present, of little patches of degeneration, and of the condition of the field of vision, as, for example, of the presence of a central scotoma, in order not to err in the diagnosis.

4. **Detachments of the Retina** that are situated not too far from the papilla may give rise to a confusion with a partial, or a total optic neuritis, through an accompanying œdema that reaches to the papilla, a cloudiness of the retina, and a tortuosity of the vessels, but this error also may be avoided by an examination of the periphery and of the field of vision (see page 154). This possibility must always be borne in mind.

5. **Commotio retinae,** when it affects the vicinity of the optic nerve, may sometimes give rise to an error (Fig. 43), at least at first glance. The absence of involvement of the vessels and the rapid disappearance of the trouble, taken together with the history, are diagnostic.

6. The **cloudiness of the region of the papilla** caused by occlusion of the central artery can give rise to this mistake only until the cherry red spot has been seen in the macula. It is associated with amaurosis, while usually there is only a great impairment of the vision in optic neuritis (Fig. **44**).

7. The **so-called supertraction crescent of myopia** (see Fig. 5 and page 35) can simulate at least a partial optic neuritis. The absence of other symptoms of this disease and the demonstration of other myopic changes, such as a conus, render the diagnosis positive.

of the chorioid that are elsewhere visible. It is not always as distinctly marked as it is in Fig. **22,** in which case unusually favorable conditions are present; the œdema is particularly great and the fundus is very rich in details, so that the contrast between the veiled and the unveiled parts is very striking. The same conditions, to a less degree, are shown in Figs. **18 20.** Sometimes it can be perceived only from the fact that a delicate veil seems to be spread over certain parts of the retinal vessels.

The margins of the optic nerve are obscured by a peripapillary œdema in optic *neuritis*, while the same symptom is produced in *pseudoneuritis* by a copious development of the supporting tissue; this is why so much stress is laid on the demonstration of œdema in the diagnosis of the former. In many cases the redness and cloudiness of the papilla, and of its vicinity, caused by this œdema is so great that both exhibit the same color, and then the difficulty of distinguishing them apart is so enormous that the position of the papilla can be recognized only from the confluence of the vessels.

Note to **3.** A distinct *elevation of the papilla* is not apt to be present in optic neuritis, but any excavation that exists may be filled up by the œdematous tissue.

Note to **4.** The *veins* alone are really changed; they are broadened and tortuous, while the arteries usually retain a normal caliber. The determination of this fact is of importance in the differentiation from choked disk, in which a marked disproportion in the fullness of the two kinds of vessels is brought about by the simultaneous diminution in the size of the arteries. The vessels are not infrequently provided with white stripes.

Patches of degeneration and hemorrhages are often found near the papilla, or in the region of the macula, yet less often than in connection with choked disk. The demonstration of such hemorrhages is likewise of the greatest importance to the diagnosis of optic neuritis as they are naturally absent in pseudoneuritis.

The vision and the condition of the visual field are also of great value. In optic neuritis the vision is apt to be very much impaired from the first, as the result of a larger or smaller, relative or absolute scotoma, while in choked disk it is apt to be normal, at least at first. In pseudoneuritis the vision is either normal, or impaired by the associated refractive error, but no anomaly is ever found in the visual field.

The **Course of Optic Neuritis.** The onset of optic neuritis may be acute and may, in that case, become fully developed in a few days, or it may take a chronic form, when the inflammatory lesion may affect only a portion of the papilla, for example, its nasal half, and thence may gradually spread to the whole.

A better delimitation of the papilla reappears with the subsidence of the inflammatory symptoms, the hyperæmia dies away, and only the change in the vessels and a smoky cloudiness over the papilla and its vicinity, through which the former appears of a dull red yellow, remain as mementos of the

presence of a morbid process (Fig. **21**). The functions return to a greater or less degree with the involution of the inflammatory symptoms, but, even in the cases in which the vision and the visual field become quite normal again, the papilla finally shows a whitish, atrophic discoloration, which may be either total or partial. In the cases in which this discoloration is scarcely suggested there is still a change in the tone of color of the papilla from the soft, delicate, peachlike hue, to a hard, porcelainlike red, which is more marked on the nasal than on the temporal side, where the atrophy is always the more distinct. A complete white discoloration of the optic nerve naturally appears in the course of time in the cases in which a greater functional trouble results. This has already been described on page 57.

## What Etiological Conclusions can be Drawn from the Ophthalmoscopic Picture of an Optic Neuritis?

It can be seen in Figs. **18 22** that, in spite of the uniformity in the essential points, the various pictures present considerable differences, and the question arises whether these differences may not be utilized in ascertaining the etiology. In regard to this it must be noted that there are no quite characteristic differences, but that certain points may be utilized in one way or another.

It has already been mentioned that the forms of optic neuritis caused by general diseases are for the most part bilateral, while those due to local lesions are usually unilateral. Although this statement does not hold good absolutely, yet it is a guide to a certain extent. It cannot be emphasized too strongly that we must not be content with the diagnosis "optic neuritis" in our ophthalmoscopic examination, but must carefully investigate the other parts of the eye in search of points indicative of the etiology of the disease of the optic nerve. If we should fail to find the hemorrhages and white spots characteristic of the albuminuric form, which have been described on page 133, we have to notice whether patches can be found in the chorioid that suggest by their appearance a syphilitic, or a tuberculous origin. Very marked obscuration of the margins of the nerve is suggestive of syphilis.

Further points may be drawn from the following:

1. *Syphilitic optic neuritis* (neuro-retinitis specifica) can often be recognized from the presence of a large œdema, which extends into the retina for the distance of 2 papillary diameters and may be so dense as to cover the optic nerve and the retina with so uniform a layer as to render it impossible to tell where the former stops and the latter begins. In such a case the situation of the papilla can be determined only from the confluence of the vessels. Usually there is a diffuse, central opacity of the vitreous which contributes to render the picture still more obscure.

Old or fresh patches in the chorioid, with deposits of pigment, are frequently to be seen in the periphery. The retinal vessels also show sclerotic changes in many cases, in consequence of which hemorrhages, arranged like

the spokes of a wheel, and white spots are not uncommon, though much less often present than in albuminuric retinitis. If the picture presents this form we may be quite certain that the case is one of syphilis. Only too often we do not find these distinctive features, but see such a picture as that shown in Fig. **18.**

The extent of the œdema depends in part on from what place in the optic nerve the inflammation starts. It may originate either from the sheath of the optic nerve, or from its interstitial tissue. In the former case we see the large œdema described above (perineuritis), in the latter the form illustrated in Fig. **18** (interstitial neuritis).

**As Wassermann's test must be made in every case,** it is rarely that the diagnosis of syphilitic optic neuritis will fail to be made.

It should also be mentioned that a specific basilar meningitis may pass from the meninges to the optic nerve and create the picture of an optic neuritis. In these cases we have to expect, in addition to other cerebral symptoms, pareses of the ocular muscles, which give rise to diplopia, paresis of the accommodation, mydriasis, and immobility of the pupil to light, especially on one side.

An optic neuritis due to hereditary syphilis is rarely seen, and is then usually associated with meningitis. As this disease occupies the attention at the time of the acute symptoms, the inflammation is observed only in exceptional cases, usually the atrophy alone is to be seen (Fig. **58**).

2. As a rule it is not possible to recognize *tuberculous* optic neuritis as such from the ophthalmoscopic picture, unless tubercles are visible at the time in the chorioid or the optic nerve, as in Fig. **19.** It is met with almost always in children, or in young people, and is to be diagnosed by the exclusion of every other cause that may produce an optic neuritis and the existence of tuberculous lesions elsewhere in the body.

The prognosis is doubtful; it is bad as regards life in the cases in which a tubercle is situated in the optic nerve (see Fig. **19**).

Although the primary inflammation of the optic nerve is the rule in *syphilis*, and the extension from the meninges forms the exception, the reverse is true in tuberculosis; the primary inflammation is very rare and the transmitted very common. *Uhthoff* says that it is the most frequent ocular symptom in tuberculous meningitis, as it occurs in from 25 to 30% of all cases.

In spite of this an atrophy as the result of an optic neuritis due to a tuberculous meningitis is seen very seldom, because most of the patients die at an early period. When an atrophy is found as the consequence of a "meningitic" optic neuritis, the cause of the latter was probably a syphilitic meningitis, as this is not as fatal.

3. *Albuminuric* and *diabetic* optic neuritis can often be distinguished by the early appearance of hemorrhages and patches of degeneration in the retina (Fig. **20**). Usually the retina is so much the more affected that the neuritis retires to the background (see Fig. **35**). In many cases an albuminuric

optic neuritis may have throughout the appearance of a choked disk (see page 76).

A point in the differential diagnosis of diabetic optic neuritis is that the accompanying central scotoma is often extremely minute.

4. *Arteriosclerotic* optic neuritis is characterized by a rather sluggish course and more or less distinct arteriosclerotic changes in the vessels of the retina (Fig. **21**).

To make the diagnosis of arteriosclerotic optic neuritis from such a picture is permissible only when the morbid process is observed from the start,
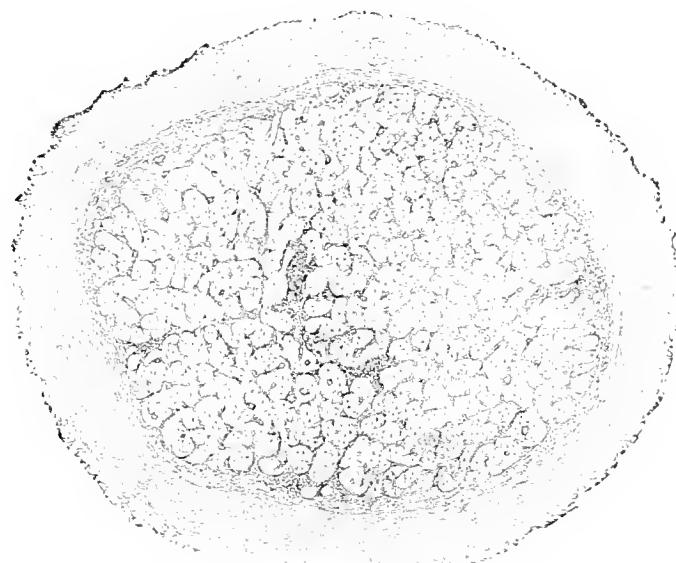


Fig. O.—Neuritis Optica.

Picture showing the pathology of the inflamed optic nerve, from *Roemer's* "Textbook of Ophthalmology." The interspaces between the bundles of fibers are dilated, and the fibers themselves, as well as the interstitial tissue, are permeated with numerous lymphocytes.

and it has been ascertained that this picture represents the acme of the disease; otherwise the same picture could be brought about by an optic neuritis due to some other cause which was undergoing involution. In exceptional cases an arteriosclerotic optic neuritis may also present the appearance of a choked disk.

5. The *otogenous* optic neuritis usually exhibits only engorgement and hyperæmia; the vessels are changed only a little, and there is little œdema. But, if a complication is added to the otitis in the form of a meningitis, an abscess, or a sinus thrombosis, the œdema increases very rapidly and such a picture may ensue as that shown in Fig. **22**, while it is not necessary that the functions be materially altered. It has frequently been observed that the inflammatory symptoms augment considerably after the opening of an ab-

scess, for example, but this has no unfavorable influence on the prognosis. Under proper treatment it usually runs a benign course.

6. The forms of optic neuritis that are caused by *abscesses* in the *orbit* and *empyemas* of the *accessory sinuses* show a marked contrast to the otogenous in that the disturbance of vision, a central scotoma which is often very large, may be quite considerable at a time when scarcely anything wrong can be seen on the optic nerve. The prognosis is usually good when the diseased cavities are opened at the proper time, yet it must be made with some reservation. Sometimes these forms of optic neuritis resemble in their course the axial, which is described below, sometimes a marked choked disk. In many cases of abscess of the orbit a thrombosis of the retinal vessels is produced, which can be recognized by the deep black color of the columns of blood and the absence of the pressure pulse (see page 107).

7. *Sympathetic* optic neuritis is a very rare phenomenon, but when it is met with it is usually in company with the characteristic roundish patches in the chorioid illustrated in Fig. **40.**

8. No *other forms* of optic neuritis have any distinctive characteristics; it is necessary to rely wholly on the results of the general examination in order to determine their etiology.

## III. Axial Optic Neuritis [1]

(Neuritis fasciculi papillomacularis; Toxic neuritis; Retrobulbar neuritis.)

An ophthalmoscopic picture of this condition is not presented in the atlas, because no change is produced in the appearance of the head of the optic nerve in 95% of the cases of the disease in question, and in the remaining 5% the only change is a little hyperæmia and engorgement, at least in the chronic cases. In acute poisoning with methyl alcohol more marked symptoms are to be seen, some resembling a partial atrophy, some a choked disk. Individual cases present an atrophy of the head of the optic nerve that is demonstrable on the third day. The diagnosis is possible in all chronic cases only through the demonstration of certain subjective symptoms.

The disease usually begins suddenly with a visual disturbance which permits the patient to orientate, but precludes the distinct perception of fixed objects, and especially renders it impossible for him to read. If he is then examined with the perimeter, or with *Haitz'* charts, the demonstration of a central scotoma is fairly easy. The examination with colors is particularly important; the color of small green and red objects cannot be recognized centrally, but they appear to be gray or "dark," while it is perceived at once by peripheral vision, i.e., as soon as the patient looks to one side of the object. The

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[1] The neuritis may be considered to be secondary, or ascending in this disease, the origin of which is a destruction of the ganglion cells of the retina, as has been proved by the pathological examination of persons poisoned with methyl alcohol during the past year in Berlin.

perception of yellow and of blue is also lost in the later stages, but the demonstration of a central scotoma for green and red in the beginning is decisive as regards the diagnosis. The size of the central scotoma is not absolutely dependent on the extent of the area of distribution of the papillomacular bundle: it may be smaller than this, as in diabetes, or it may be considerably larger when the adjacent parts of the optic nerve are involved, especially in diseases of the accessory sinuses. At first there is usually nothing to be seen with the ophthalmoscope: it is not until the process passes over into atrophy that a paleness of the temporal side of the optic nerve is to be seen (see page 58).

The same diseases are to be taken into account **etiologically,** as in partial, or temporal, atrophy of the optic nerve, diseases of the accessory sinuses, intoxications, and multiple sclerosis (see page 59).

It should be remembered that a number of diseases, such as empyema of the accessory sinuses, lead poisoning, and diabetes, may induce the picture of either true optic neuritis, or of axial optic neuritis.

The **prognosis** depends on the cause and the stage of the disease.

When it is possible to induce an alcoholic to abstain from liquor his vision may return, otherwise his optic nerves will become permanently atrophic. The prognosis is not so good, though it is not absolutely bad, in cases of poisoning with sulphuretted hydrogen and carbonic oxide: it is rather better in diabetes.

*Note.*—The charts devised by *Haitz* are extremely useful for the demonstration of a central scotoma. These charts consist of symmetrical halves with graduated lines and can be used in an ordinary stereoscope. The two halves of the chart are superimposed by the action of prisms so that they fuse into a stereoscopic picture and appear as one. This enables the healthy eye to maintain an accurate fixation while the other is tested for the presence of a central scotoma.

## IV. Choked Disk

There is as yet no universally accepted theory as to the nature of the origin of choked disk, it is still uncertain whether it is caused purely by engorgement, or by inflammation.

This question is of comparatively little importance to the clinical picture of at least one form, the so-called albuminuric choked disk. It deserves to be particularly mentioned, on account of its great etiological importance, that in exceptional cases albuminuric neuritis may assume a form that can scarcely be differentiated from a choked disk with patches of degeneration. Arteriosclerotic optic neuritis also has the appearance of a choked disk in many cases.

The most essential points of difference between an optic neuritis and a choked disk consist in:

1. The behavior of the vessels. In choked disk there is a very considerable difference in the fullness of the veins and of the arteries; the veins

are distended, the arteries contracted, while in optic neuritis the arteries are almost normal and the veins overfilled.

2. The elevation of the head of the optic nerve.[1]   A choked disk is accustomed to rise more than 1 mm, 3 D, above the level of the retina, while a disk that is the seat of a neuritis seldom reaches such a height.

3. The behavior of the vision.

In optic neuritis the vision is usually much impaired at a very early stage (central scotoma), while in choked disk it may remain nearly, or quite, normal for a long time.

The **course of a choked disk** is as follows, according to *v. Michel:*

At first the arteries are seen to become small and to be provided with broad reflex stripes upon the papilla.   The large venous trunks are much broadened, tortuous, of a dark red color, and are destitute of pulsation. The smaller veins become more distinct because of their greater fullness. The vessels in general, but particularly the veins, appear to be bent and broken on the other side of the margin of the papilla; a large number of small vessels very often become visible on the papilla itself (Fig. **25**), and give it a reddish gray tone of color that often inclines to violet.   The papilla forms a marked elevation with a precipitous descent to the retina, and exhibits an increasing opacity with radiating lines, which covers its margins, extends out beyond them, and is bordered by a gray edge.   The excavation may persist for a while (Fig. **24**), or only a part of the papilla may be affected (Fig. **23**).   In its further course the elevation and swelling of the papilla increases, the retina in its immediate neighborhood becomes more and more opaque, and consequently gives the impression that the papilla has become broader, because as the result of its indistinct contour its margins seem to lie where the opacity ceases.   The opacity of the papilla and of its immediate vicinity exhibits more and more a striated and reddish white appearance corresponding to the normal course of the bundles of nerve fibers in the retina. Often the vessels can scarcely be perceived in the center of the papilla, but come into view first in its periphery, or at the margin of the opacity, from the swollen tissue.   The arteries appear to be still more contracted than at first, drawn out and pale, the veins, beginning with pale, pointed ends, show a deep, dark red color, have diameters that vary a great deal according to the depth at which they are situated, and bend about with great windings in the plane of the retina, in which they run tortuous courses.   Frequently the vessels are hidden, or obscured, for a distance by a gray opacity, and hemorrhages are often found arranged in radial striæ, usually in the retina at the margin of the papilla, as well as here and there in the latter itself.   Fine, brilliant white lines, ordinarily arranged radially, on and also outside of the papilla, or small, brilliant white spots, which appear at a very early period, are chiefly to be observed in children or young people.   These lines and spots often extend beyond the margin of the papilla and maintain such

[1] Concerning the way to estimate differences of level, see page 9.

an extent and grouping that the retina may present the same condition as in albuminuric retinitis. Sometimes the veins are accompanied by white stripes.

Gradually the papilla loses its reddish tone of color, which is replaced by a white, or yellowish white opacity, inclining to gray, but its margins remain obscured and the swelling continues to be plainly demonstrable. The onset of these changes ushers in the so-called atrophic stage (Fig. **13**) of choked disk, in which the protrusion of the papilla subsides. The opacity and swelling do not undergo complete involution, the arteries remain small, the veins engorged. Just as only one half, or one sector, may be swollen at first (Fig. **23**), so in the involution of the swelling the subsidence may take place in the same way. Not infrequently the pigment epithelium in the region of the opaque margin of the optic disk is decolorized.

## What Etiological Conclusions can be Drawn from the Ophthalmoscopic Picture of Choked Disk?

*Unilateral Choked Disk*

occurs in affections of the *orbit*, such as tumor, abscess, cysticercus, and gumma, and in diseases of the *accessory sinuses*. It must be remembered that tumors or abscesses in the middle fossa of the skull may protrude into the orbit.

*Bilateral Choked Disk*

occurs in all conditions of the brain that reduce the amount of space in the cranial cavity. Chief among these are all kinds of *tumors of the brain*, including not only the true tumors, but also cysticerci, aneurysms, gummata, and tubercles (about 70 to 80%). Choked disk is absent in only from 5 to 10% of the cases of tumor of the brain, and these are mainly tumors of the frontal brain and of the hypophysis. The farther back the tumor lies the more certain is a choked disk to appear. A very rapid onset of visual disturbance, with a high degree of choked disk and severe pains in the back of the head, is indicative of a gumma in the cerebellum; a choked disk with disturbances of the auditory and facial nerves, of a tumor in the angle between the cerebellum and the pons. A disturbance in the field of vision, like a hemianopsia, frequently gives an indication as to the situation of the tumor. A choked disk with horizontal hemianopsia may be caused by a hydrocephalus internus with a bulging outward of the recessus infundibuli and pressure of the same upon the chiasm. A localization of a brain tumor from the greater development of the choked disk in one eye or the other cannot be made, but it may perhaps be possible from the accompanying faults in the field of vision, such as a homonymous hemianopsia.

The cause of choked disk next in importance to tumor of the brain is serous *meningitis*, or *hydrocephalus internus*. None of the other causes, such

as oxycephalia, abscess of the brain, sinus thrombosis, and hemorrhagic pachymeningitis, are of equal consequence. In the disease last mentioned the choked disk is sometimes unilateral and associated with dilatation of the pupil.

The affections of the optic nerve observed in hemorrhages into the subdural, or subarachnoidal space, do not correspond entirely with the picture of choked disk, inasmuch as the disproportion between the arteries and the veins is not apt to be so marked. The affection is usually more pronounced on the side of the lesion than on the other.

Finally, a choked disk may result from an obstruction to the outflow of
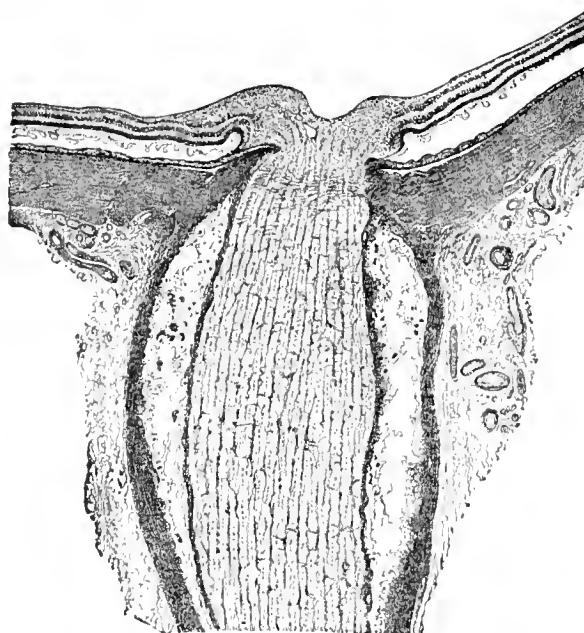


FIG. P.—Choked Disk.

In this picture, taken from the textbook by *Roemer*, the mushroomlike elevation of the papilla into the vitreous, and the great distention of the sheath of the optic nerve, are to be seen very distinctly.

the venous blood into the cavernous sinus. Concerning the thrombosis of the vessels of the retina see pages 78 and 102. Such a cause is supposed to be present in the choked disk of chlorosis.

In conclusion it remains to be said that albuminuric and arteriosclerotic optic neuritis may present the picture of choked disk, in consequence of the engorgement that takes place at the same time. *Therefore the urine should be examined and Wassermann's test be made in every case.*

The *vision* may remain normal for a long time, and therefore be at variance with the great ophthalmoscopic changes. When the choked disk passes into the atrophic stage the vision gradually disappears, but from the first the patients are tormented by temporary, fleeting attacks of blindness, or of obscuration.

The *field of vision* shows various forms of contraction. The defects may be peripheral, or in the form of sectors, or of hemianopsia, but such a central scotoma as accompanies optic neuritis is almost never seen.

### Tumors

are rarely met with on the papilla; the most common are gummata and tubercles. A conglomerate tubercle is shown in Fig. **19.**

Sometimes developments of connective tissue are seen to extend out from the papilla; these may be the remains of fetal structures, the results of injuries, or the products of organization of hemorrhages.

### Hemorrhages on the Papilla

may appear in optic neuritis, or in choked disk, or when the vessels of the retina are sclerotic. Sometimes they result from injuries to the eye, or to the optic nerve.

The demonstration of a hemorrhage, when it is not of traumatic origin, is always of great diagnostic importance. For example, it immediately decides the question in a doubtful case of optic neuritis or pseudoneuritis in favor of the former (see page 71).

### Wounds of the Optic Nerve

in the *orbit* produce an ophthalmoscopic picture that varies accordingly as the nerve is severed in the portion that contains the vessels, or in the part that does not. In the former cases the signs of an occlusion of the central artery are present (see Fig. **44,** and page 150), in the latter case the optic nerve appears to be perfectly normal in spite of the blindness, until atrophy gradually develops, in the course of about 6 weeks (see Fig. **10,** and page 54).

PLATE X

Fig. 18. Optic Neuritis

Fig. 19. Tubercle at the Entrance of the Optic Nerve

# Fig. 18.   Optic Neuritis

The margins of the optic disk are quite indistinct, the disk itself is much reddened and is surrounded by a gray areola, due to œdema. The veins are much distended and are slightly hazy in the gray zone caused by the retinal œdema. They are accompanied by whitish stripes on the papilla. A slight elevation of the disk can be made out by parallactic displacement, or by determining the refraction with the ophthalmoscope. The condition was due, in this case, to syphilis.

The gray ring about the papilla is of special value in the diagnosis (see page 72).

# Fig. 19.   Tubercle at the Entrance of the Optic Nerve

The larger part of the papilla is obliterated, its margin on one side is completely hidden. At that place is a whitish mass, nearly as large as the papilla, which is shown by parallactic displacement to be distinctly elevated. It is surrounded by a slightly gray discoloration of the fundus, caused by an œdema of the retina. The retinal vessels that end at this place plunge into the mass and their terminal portions are invisible. It is striking that no superior temporal artery can be seen. The fundus is of the albinotic type and is normal, except for the gray discoloration in the vicinity of the lesion; the retinal vessels elsewhere are normal.

The diagnosis in this case was that of a tumor at the entrance of the optic nerve. The specific diagnosis of a tubercle, or of a conglomerate of tubercles, in the head of the optic nerve, was based upon the local reaction that followed an injection of tuberculin.

Tab. 10.
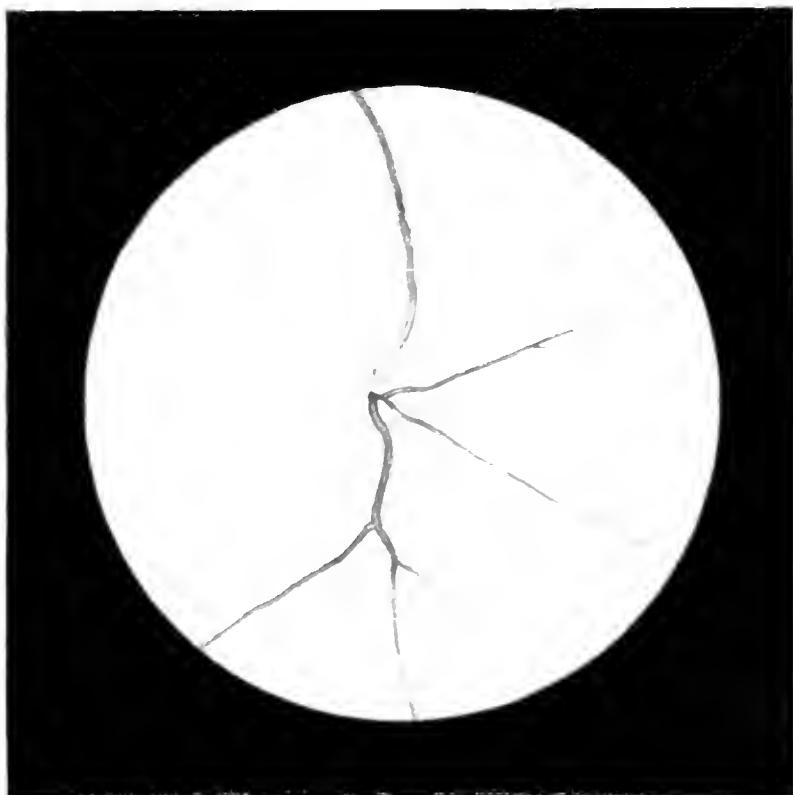


Fig. 18.
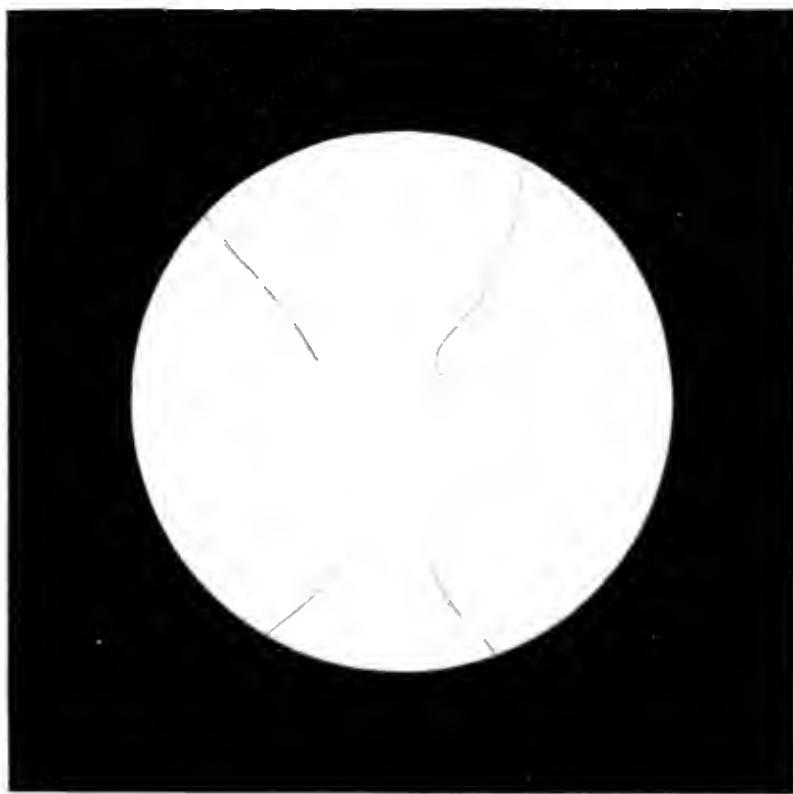


Fig. 19.

# PLATE XI

Fig. 20.  Albuminuric Optic Neuritis (Albuminuric Choked Disk)

Fig. 21.  Optic Neuritis Undergoing Involution

## Fig. 20. Albuminuric Optic Neuritis (Albuminuric Choked Disk)

(See page 76)

The papilla is very red and swollen, its margins are obliterated, its arteries reduced in size. It was mistaken at first for a choked disk caused by a tumor of the brain, but, as the result of the neurological examination was negative, and the urine presented the characteristics of chronic nephritis, that diagnosis had to be abandoned and replaced by that of an albuminuric neuritis (see page 79). At some distance from the papilla may be seen hemorrhages placed radially, therefore superficial, and some stipplings that indicate patches of degeneration on the temporal side. These patches of degeneration are not inconsistent with a typical choked disk, for they are sometimes found in association with it, but they are to be seen more commonly with neuroretinitis albuminurica.

The fundus, which is on the whole of the uniform, stippled type, approaches the tesselated in its nasal portion.

The absence of the stellate figure in the macula is in no way contraindicative of albuminuria, for it is present in by no means all cases of albuminuric diseases of the retina.

## Fig. 21.—Optic Neuritis Undergoing Involution

(See pages 74 and 76)

The margins of the papilla have already become rather more distinct. The color is a cold red in the center, while the marginal portions are paler. The vessels are distinctly sheathed. Pigment has migrated into the retina around the optic nerve, which accounts for the dark gray discoloration.

Such a picture as this may also be indicative of the so-called arteriosclerotic optic neuritis, a form of inflammation of the optic nerve that is met with in old people, and is characterized by a very sluggish course of little intensity, in which the changes in the retinal vessels stand in the foreground.

Tab. **11.**
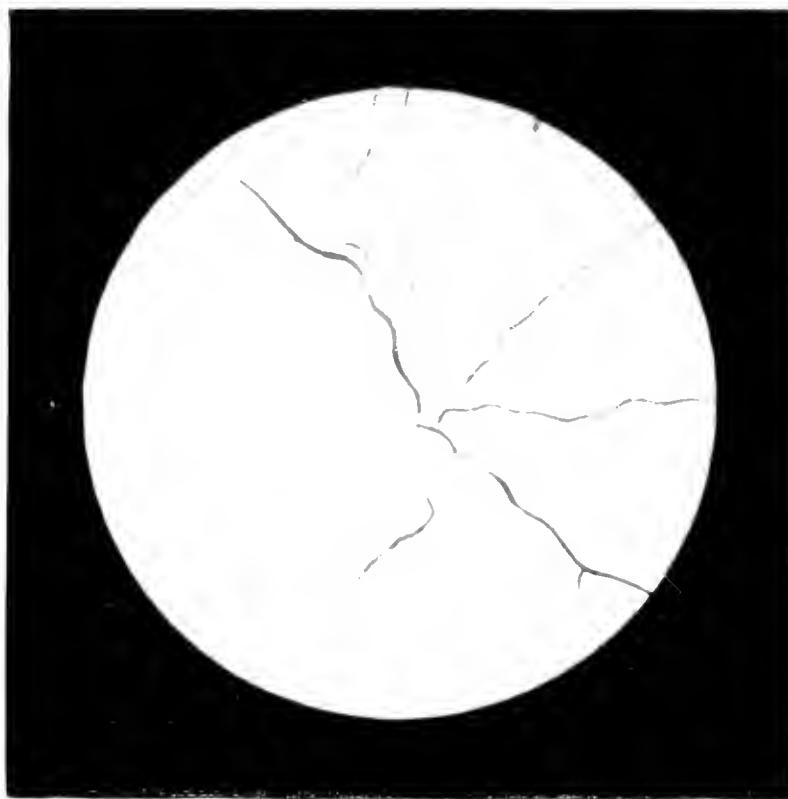


Fig. 20.
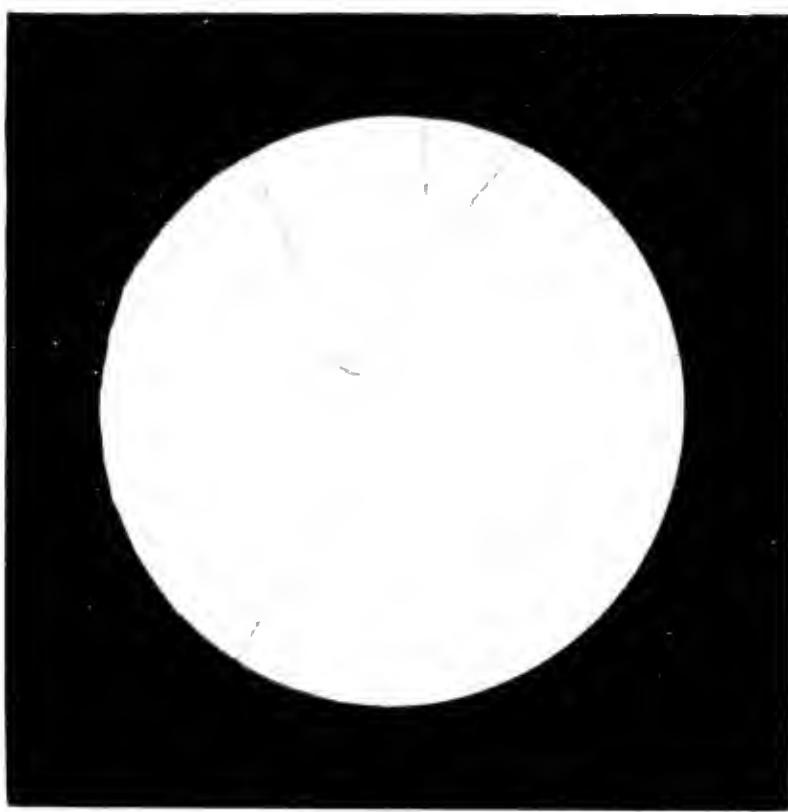


Fig. 21.

PLATE XII

Fig. 22. The Optic Nerve in a Case of Sinus Thrombosis Complicating an Otitis Media

## Fig. 22. The Optic Nerve in a Case of Sinus Thrombosis Complicating an Otitis Media

(See page 77)

A slight hyperæmia of the papilla is sometimes seen in uncomplicated cases of otitis media, but as soon as a cerebral complication takes place, such as a sinus thrombosis, an extradural abscess, or a meningitis, the redness of the papilla becomes greater without causing the margins to become particularly indistinct. The veins show only a trifling congestion. What is specially noticeable is the enormous œdema of the retina which surrounds the head of the optic nerve and hides everything that lies beneath it. The retinal vessels may rise through the œdema and show a distinct parallactic displacement; when they are placed more deeply they are partially covered by the œdema, as is the case with the vessel situated above in the picture.

The œdema is very clearly visible in this case because it is quite extensive, and because the almost albinotic fundus shows up many details that are lacking in the œdematous portion. The œdema cannot be seen as well when the color of the fundus is uniform.

As soon as changes that are to some degree distinct have appeared in the head of the optic nerve it becomes the duty of the aural surgeon to open the skull. This operation is sometimes followed by an exacerbation of the intraocular condition, but the prognosis of this is *not* bad.

Tab. 12.
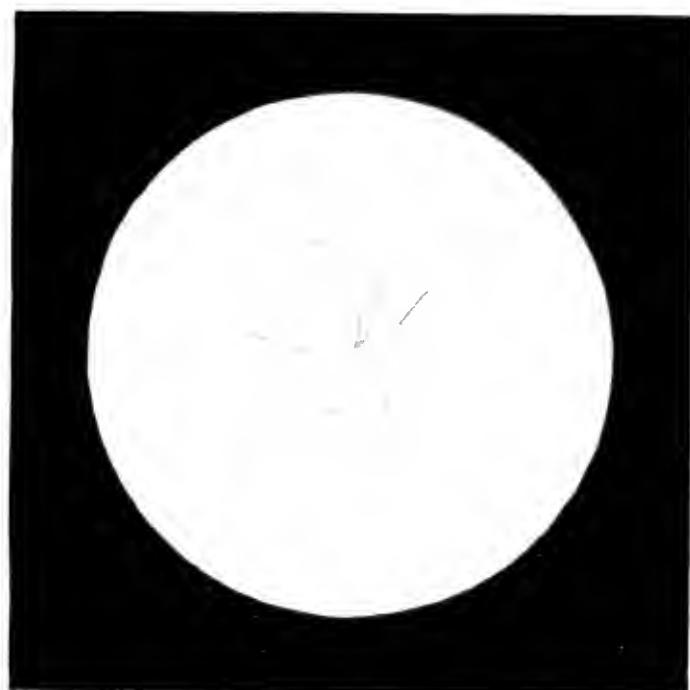


Fig. 22.

# PLATE XIII

## Fig. 23.   Commencing Choked Disk

It is important to be able to recognize the early stage of a choked disk on account of its significance in diagnosis.

It can be seen from the course of the vessels that the temporal portion of the papilla, where the nerve fibers are feebler, has not yet been driven forward, while the nasal is distinctly elevated. The œdema that causes the elevation has already invaded the vicinity so as to make the papilla appear to be enlarged and to completely obscure its margins. The veins are distended, while only a part of the arteries show that their caliber is diminished.

## Fig. 24.—Commencing Choked Disk

This picture shows another form. The entire periphery is protruded, while the center still remains at its old level. The disproportion between the arteries and the veins is clearly marked, as well as the œdema that surrounds the papilla.

Tab. **13.**
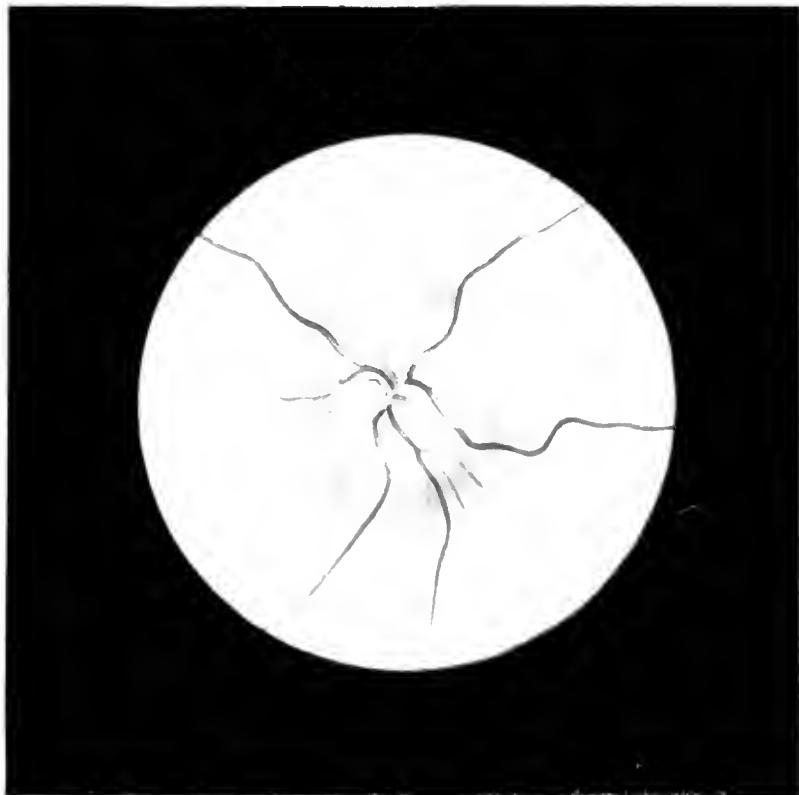


Fig. 23.
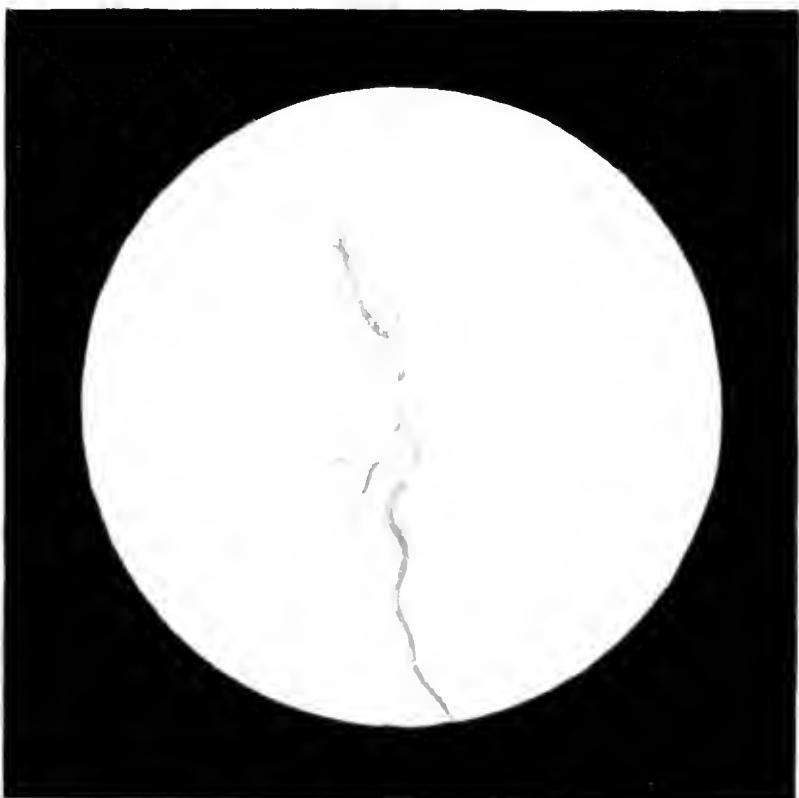


Fig. 24.

PLATE XIV

Fig. 25.  Old Choked Disk with a Very Abundant Development
of Vessels

Fig. 26.  Choked Disk at Its Acme

## Fig. 25.   Old Choked Disk with a Very Abundant Development of Vessels

(See page 81)

Two forms of choked disk can be distinguished in well-marked cases, one distinctly knob-shaped, the other more diffuse. The former is shown in this picture.

The delimitation of the swollen portion from the retina is comparatively distinct, although the protrusion is considerable, as may be seen from the course of the vessels, and œdema is also certainly present. The disproportion between the arteries and the veins forms the principal ground for the diagnosis.

On the papilla are to be seen a large number of newly formed vessels; this indicates that the choked disk has lasted a long time.

The other papilla of the same patient, which was distinctly atrophic, is shown in Fig. **22**.

The lesion in this case was a gliosarcoma of the cerebellum.

## Fig. 26.   Choked Disk at Its Acme

(See page 80)

The more diffuse form is shown in this picture. Scarcely a trace can be seen of the margins of the papilla, but the latter seems to send tonguelike processes into the retina. The papilla is distinctly elevated, as can be seen from the course of the vessels, and exhibits a radiating striation. A number of hemorrhages, also striate in form, give the disk quite a specific appearance. The disproportion between the arteries and the veins is so great at the acme that the former are scarcely visible, while the latter leave the papilla as broad, tortuous bands. Some white patches of degeneration are visible in the retina. There are only a few retinal hemorrhages in this case, but they are often much more numerous.

The vision in this patient was normal. The neurological examination revealed the existence of a tumor in the angle between the cerebellum and the pons, the so-called acusticus tumor.

Tab. 14.
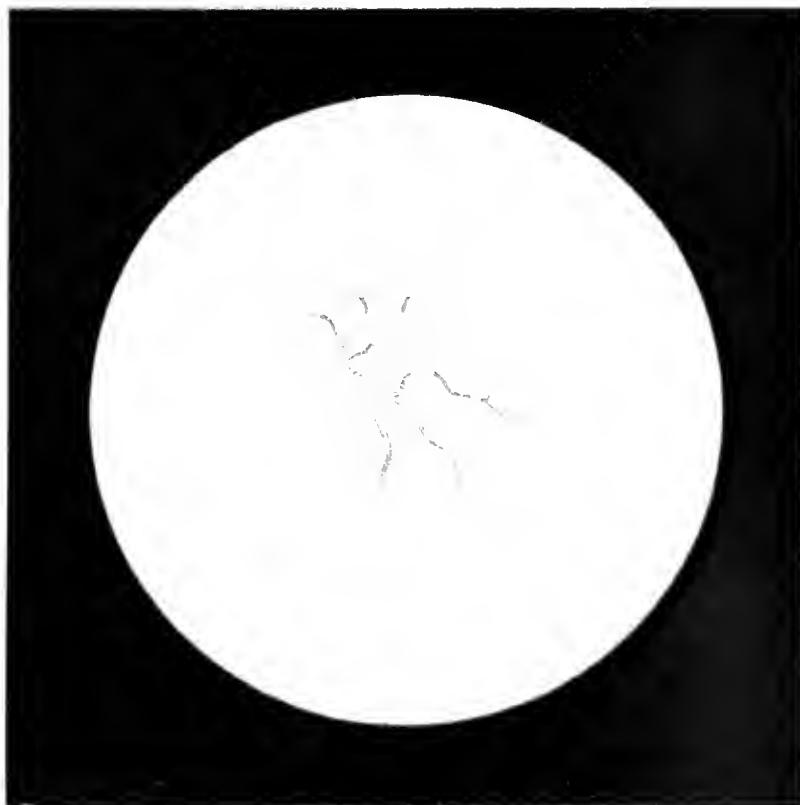


Fig. 25.
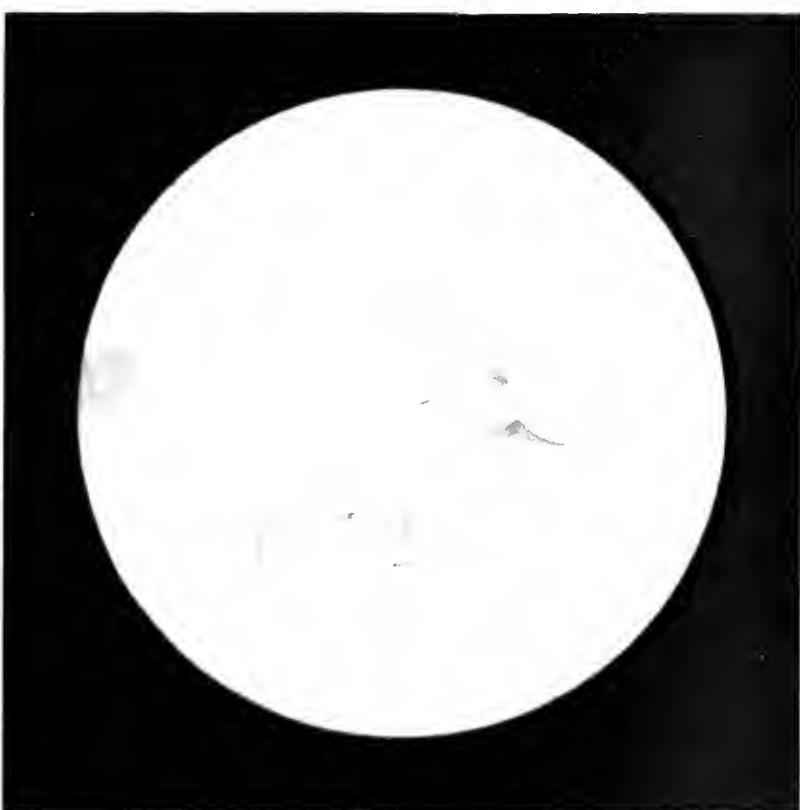


Fig. 26.

Vessels of the Retina

# Vessels of the Retina

The great diagnostic importance of changes in the vessels of the retina has already been pointed out in the study of the diseases of the optic nerve. Nowhere else in the body can the blood vessels be seen so clearly, nowhere else are they so accessible to direct observation, nowhere else is our duty so imperative to study the minutest details of the picture, as in the vessels of the retina, the more so that they are offshoots from the vessels of the brain, and that certain conclusions can be drawn from their condition with reference to that of the cerebral vessels. *Bouchut* termed ophthalmoscopy direct cerebroscopy, and, although this expression overshoots the mark, it indicates how highly ophthalmoscopy is to be valued (compare page 153).

A special chapter is devoted here to changes in these vessels in order to indicate their importance in the most forceful manner, and to lead to their careful observation.

In many cases the *sequela* of diseases of the vessels stand forth in such a manner, as, for example, in Fig. **44**, that a certain schooling is needed to think of, and correspondingly to investigate, the cause of such changes: in others, as in Fig. **28**, the changes in the vessels themselves are so prominent that they explain the clinical picture. *We should therefore not be content to cast a brief glance at the papilla in an ophthalmoscopic examination, but should accustom ourselves to investigate the vessels of the retina very thoroughly.*

The changes relate chiefly to the caliber and walls of the vessels, more rarely to their contents, and, under certain circumstances, pulsatory phenomena are to be taken into account diagnostically.

## Preliminary Remarks on the Anatomy

It must be remembered that the walls of the retinal vessels are perfectly transparent, and therefore invisible, when they are in a normal condition; also that under some pathological conditions, as, for example, in hyaline degeneration, they may be perfectly transparent in spite of a considerable thickening that is made evident only by a narrowing of the blood column. Thus what is seen ophthalmoscopically is not the entire vessel, but only its contents; the wall has to be added mentally, so that the vessel is really twice as thick as it appears to be in the ophthalmoscopic picture.

On the other hand, defects appear in the transparency of the vessel wall which may be observed in all degrees, from a scarcely perceptible veiling to a

complete opacity. The color assumed by the vessel may then be a pure white, a gray white, a yellow white, or a light brown; both the opacity and the reflection may be fairly variable.

In the observation of the transverse diameter of the vessel the observer is subject to many illusions, which are due not only to the optical conditions present in the eye, but also to the more or less deep situation of the vessel in the retina. The apparent size of the papilla must serve as the unit of measure.

## The Changes in the Vessels of the Retina

take place in

 *A*. The **caliber:** Differentiation must be made between

  1, *Contractions* up to total disappearance of the vessels,

  2, *Dilatations,*

  3, *Differences* in the *proportionate caliber* of the arteries and of the veins,

  4, *Unevennesses* in the *caliber* of individual vessels.

 *B.* The **color:** The change of color may be due to

  (*a*) the color of the *blood column,*

  (*b*) the color of the *wall of the vessel;* in regard to this are to be noted

   1, accompanying stripes,

   2, transformation into white cords,

   3, infiltrates in or over the vessels.

 *C.* The **number** and **arrangement**.

 *D.* The **course;** drawn out, tortuous, wavy, broken.

 *E.* The **reflex;** its breadth and intensity.

 *F.* The **phenomena** of **pulsation.**

# ELABORATION OF THE ABOVE SUMMARY

## A. The Caliber

1. *Contraction.*

A change in the length of a vessel is usually associated with a decrease of its transverse diameter; at first it is drawn out, later it is distinctly shortened. The reflex and the color are also apt to be changed.

Four forms are to be distinguished etiologically:

(*a*) That which is produced pathologically, commonly termed sclerosis;

(*b*) that which is functional, caused by contraction of the muscular tissue in the wall of the vessel;

(*c*) that which is due to compression, the cause of which is to be sought in a compression of the afferent vessels;

(*d*) that which is due to an imperfect filling of the vessels with blood.

The first form, sclerosis, is the most common. As appears from the above remarks on the anatomy, the contraction is usually only an apparent one; of such a nature that the transparent, and therefore invisible, wall of the vessel is thickened so as to render the blood column within it smaller, but true diminutions and atrophies of the vessels themselves are met with. It is not usually possible to distinguish the two forms ophthalmoscopically.

As considerable differences occur normally in the caliber of the vessels in different persons, and as the vessels in the same person vary in size one from another, it is often quite difficult to determine whether a commencing sclerosis is present or not. In such doubtful cases attention has to be paid to fluctuations in caliber,[1] to abnormally distinct pulsation, and to the sheathings that may perhaps be present. These conditions are almost always to be held to indicate a commencing sclerosis of the vessels, the extremely rare congenital sheathings being excluded.

If the sclerosis is more advanced, for example, as in Fig. **67**, no further difficulties are encountered in its diagnosis. As may be seen in Fig. **56**, the change may go so far as to render the vessels no longer visible.

The changes may take place in both the arteries and the veins, either independently of each other, or together. In the latter case the physiological difference in the breadth of the veins and the arteries is maintained to the last, so that when the vessels disappear the arteries are the first to become invisible. In the majority of cases the arteries alone are affected, while the veins at first seem to be rather broadened on account of the elasticity of their walls.

As complications, or sequelæ, may be named atrophy of the optic nerve (see page 53 and Fig. **6**), acute occlusion of the arteries with its consequences (see page 150 and Fig. **44**), thrombosis of the veins (see page 117 and Fig. **27**), and extensive disease of the retina in the form of retinitis albuminurica (see page 133 and Fig. **31**), or diabetica (see page 134 and Fig. **35**).

*Etiologically,* the first disease to be taken into account is syphilis, either acquired (Fig. **38**) or hereditary (Fig. **56**), then arteriosclerosis (Fig. **67**), chronic nephritis and diabetes. The high degree of contraction of the vessels found in retinitis pigmentosa (Fig. **51**), which affects both the arteries and the veins, depends on similar causes to all appearance. Contraction of the arteries alone is to be observed as a sequel of an old occlusion (Fig. **16**), and sometimes also in old cases of glaucoma.

A uniform functional contraction of both the arteries and the veins is found in cases of poisoning with quinine or ergotin, in chronic alcoholism and in commencing syncope.

A *compression* of the arteries is associated regularly with a compression of the veins, but while it shows itself in the arteries by a diminution of the blood column due to an insufficient supply of blood, it causes an engorgement

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[1] The apparent diminution of the vessel to a point at the place where it leaves the papilla is not to be understood as a fluctuation in caliber in this sense.

of the veins by obstructing the escape of the blood. We meet with compression chiefly in cases of tumor of the orbit and affections that reduce the space within the skull. In the former it is unilateral, in the latter bilateral. As the venous engorgement forms the most conspicuous part of the picture, this condition will be described below, under Dilatation of the Vessels.

A contraction of the arteries takes place in cases in which there has been a *great loss of blood*, as in labor, from ulcer of the stomach and from wounds, but this usually passes off very quickly after the hemorrhage has been checked, although a more or less severe functional disturbance of the vision persists in many cases, which is due to a partial or total atrophy of the optic nerve.

2. *Dilatations of the Vessels.*

Just as a straightening accompanies the contraction of a vessel, so a marked tortuosity is commonly associated with its dilatation. The reflex and the color of the blood column are also accustomed to undergo a change, the former becoming broader, the latter darker (see Fig. **28**).

Both arteries and veins may be dilated at the same time, or the veins alone may be affected. A dilatation of the arteries alone is not likely to be observed. If the dilatation involves both the arteries and the veins the normal difference between their transverse diameters is maintained. The color of the fundus is not changed by the increased fullness of the vessels, on the contrary the papilla takes on a livelier color. The vessels seem to be increased in number because the smaller ones, which are usually invisible, come into view. At the same time it must be remembered that under certain circumstances the number may seem to be smaller than normal, as is the case when there is coincidently a great deal of œdema; for example, as in a neuro-retinitis; the smaller and deeper branches may then be buried completely in the œdema.

## (*a*) Uniform Dilatation of the Veins and Arteries

is met with as a local symptom of general plethora in fevers, in constitutional anomalies, such as plethora, or the apoplectic habit, and in overindulgence in alcohol.

Ocular causes to which it may be due are the sudden relaxation of tension caused by operative or accidental wounding of the eyeball, the relief from pressure, as in operations for strabismus, and as the result of local congestion produced by contusions, by a downward inclination of the head, by too much light, as when an ophthalmoscopic examination is prolonged unduly, or by too great demands having been made on the accommodation. It is likewise met with in inflammation of both the anterior and posterior segments of the eyeball. In this respect inflammation of the optic nerve is to be mentioned as of special importance (see Optic Neuritis, page 72).

It is considered by some authors to be an early symptom of disease of the accessory sinuses which may be the very first to appear. It is also to be noticed as the first sign of a cerebral complication, or of a sinus involvement, in diseases of the ear.

In leucocythæmia the veins are apt to be enormously dilated, while the dilatation of the arteries is only moderate. The color of the fundus in these cases is orange red.

## (b) Venous Hyperaemia with the Arteries Normal or Contracted

Venous hyperæmia is present in general cyanosis due to congenital, rarely to acquired, heart disease, as well as in pneumonia, emphysema, and polycythæmia.

It is due to local causes:

(α) In the early stage of phlebosclerosis; contraction ensues in the later stages.

(β) In acute glaucoma (Fig. **14**, pulsation of the arteries, varicosities of the veins), and in secondary glaucoma, for example, from an intraocular tumor (Fig. **50**).

(γ) In thrombosis of the veins (Figs. **27** and **28**), the most essential and most prominent symptom in which is the enormous hemorrhages.

(δ) In inflammation of the head of the optic nerve (albuminuric choked disk) and of the retina (Fig. **20**, page 79).

(ε) In compression of the vessels, either directly by tumors of the orbit, when it is unilateral, or by processes that increase the pressure in the brain, when it is bilateral (Fig. **26**).

The dilatation of the veins may be so considerable as to make them appear to be from one quarter to one and one half times as broad as normal.

It is to be noticed in those affections which are accompanied by an œdema of the retina that the vessels are sometimes so embedded in the œdema that only a portion of their transverse sections can be seen.

*3. The Differences in the Proportional Sizes of the Arteries and of the Veins*

have been dealt with in the preceding chapter, and the only thing necessary is to call attention again to the great importance of this point in the differential diagnosis of optic neuritis from choked disk (page 79).

*4. Unevennesses of Caliber*

in the course of individual vessels may be real or only apparent.

Real inequalities may be caused by uneven scleroses of the walls; uneven in both the sense that the sclerosis is more marked in some places than in others, and the sense that the wall of the vessel is thickened more on one

side than on the other, so that the lumen is displaced from the center of the vessel, or is made oval. As scleroses may affect the wall of the vessel without producing any opacity in it, and as the breadth of the vessel fluctuates within fairly wide limits under physiological conditions, especial weight is to be placed on *such unevennesses of caliber in the diagnosis of commencing sclerosis.*

Secondly, the inequality of caliber may lead to greater or less pouching out of the wall of the vessel, as shown in those that course upward in Fig. **36**, forming aneurysms of the arteries, phlebectasiæ of the veins.

Thrombi may readily form in the places where the vessels are pouched out.

An *apparent* change of caliber may be produced by the embedding of the vessel in the swollen or œdematous retina (see Fig. **33**), or in the tissue of the tumor (Fig. **50**).

A variation in caliber may also be simulated by the presence over the vessel of such tissues as medullated nerve fibers (Fig. **9**), bands of connective tissue (Fig. **37**), and masses of exudate (Fig. **38**). Partial interruptions in thrombosed vessels may also simulate such an appearance.

## B. The Color of the Vessels

is dependent on the color of the blood, of the vessel wall, and of its surroundings. Ceteris paribus a vessel appears to be darker on a bright than on a dark background. A vessel that is buried very deeply in the retina appears to be darker than one that is superficial.

The *vessels are pale* in anæmia, chlorosis, and, to a very marked degree, in leucocythæmia. In the latter disease the color of the veins is almost the same as that of the arteries, so that the former can be distinguished only by their greater breadth and tortuosity.

The *vessels are dark* in thromboses (Fig. **28**), and in venous engorgements of either general or local origin. In these cases the ordinary difference in the color of the arteries and the veins is particularly distinct.

In cases in which the thrombosis is secondary, for example, to an orbital cellulitis, the *vessels are very dark* and seem to be almost black.

The change of color sometimes affects only the sides of the vessels, when it produces the appearance of accompanying stripes, sometimes its entire breadth, sometimes only certain portions of it.

The **accompanying stripes** may be due to various causes. Sometimes they indicate a *commencing sclerosis;* in these cases the blood column is commonly narrowed. Although they are to be seen most plainly in the neighborhood of the papilla they are to be found there least often, because the smaller vessels are usually diseased before the larger.[1] In most cases they

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[1] The sheathings of the vessel confined exclusively to the vicinity of the papilla are usually the results of neuritic changes (see under Proliferation of glia).

spread out irregularly and may have sharply defined edges, or may blend gradually with the transparent portion of the retina. Sometimes these stripes can be seen to accompany the vessel throughout its entire length. In very marked cases the whole vessel may finally be transformed into a white cord (Fig. **56**).

The very first sign of such a disease is usually a broadening of the central, reflex light streak, after which the changes develop that have been described and the entire vessel becomes gradually transformed into a slender, white cord. Generally the color is pure white, but it may be gray, or reddish gray. The pure white color prevails in the sheathings to be described below.

Pathologically, we find cellular and connective tissue proliferations of the intima and adventitia. The latter may lead to the so-called retinitis proliferans (Fig. **37**).

The sequelæ consist of diseases of the retina and of the optic nerve, the same as in simple sclerosis, and the same etiological factors take part. As a rule the two forms of disease cannot be recognized as distinct, as the one passes over into the other; they are spoken of here under two heads simply for the sake of clearness.

Secondly, the accompanying stripes may be caused by a *proliferation of the glia, as the consequence of an optic neuritis* (Figs. **12** and **21**). It has already been mentioned that the indistinct margins and the white color of the papilla in an atrophy that results from an optic neuritis are caused by a proliferation of the interstitial glia tissue and, in harmony with such an etiology, this sort of sheathing is found only on the optic nerve, or in its immediate vicinity, or at least it is most pronounced in this locality. The demonstration of such a change is therefore valuable evidence in favor of the neuritic nature of an atrophy of the optic nerve. The color is commonly a pure white, in which it varies from other forms of sheathing.

A third form is shown in Fig. **42**. This is the one that is met with in those cases in which an acute sheathing of the retinal vessels appears simultaneously with signs of retinitis; it can be explained only as a filling of the lymph spaces of the adventitia with white blood corpuscles.

The bright bands along the upper vessels in Fig. **33** have to be explained in a similar manner, except that here the fluid has already left the sheaths of the vessels.

The white deposits of lime, and other concrements, that occur here and there, need only to be mentioned.

The complete interruption of a vessel with the signs of a sudden occlusion (see Fig. **45**) indicates the presence of an embolus, or a thrombus, within it.

Sometimes the accompanying stripes are only simulated by the reflection of light along the vessels. This sort of reflection is seen very often (see page 26), especially in young persons in whom the fundus is dark. They can be recognized from the fact that they change as the mirror is rotated. Such reflections are shown in Figs. **2** and **30**.

## C. Changes in the Number of the Vessels

The number of vessels may be diminished or increased.

A *diminution* is met with in marked sclerosis (Fig. **56**), as well as in injuries and diseases of the retina, especially those that are associated with a development of connective tissue (Fig. **36**).

An apparent diminution, due to some being rendered invisible, is to be observed when the optic nerve is suddenly severed through the portion that contains the vessels, when arteries are occluded (Figs. **44** and **45**), and when great œdema is present, as well as in cases of tumor and of detachment of the retina.

A smaller number of vessels than normal may be present congenitally, but such a condition is commonly associated with other anomalies in the fundus.

An *increase* in the number of vessels may likewise be real or only apparent.

A true increase is caused by a new formation of large veins, or of loops of small vessels. These are to be observed when gross circulatory disturbances are present in the eye, as in compression, or partial obliteration of the central vein, i.e., in such conditions as favor the origin of a choked disk, as well as in glaucoma and after thromboses of the veins. As a rule the newly formed veins are the so-called opticociliary veins (see page **23**), yet some can be seen in the retina alone.

The loops of small vessels are particularly common in choked disk (Fig. **25**): they are to be seen more rarely in diseases of the retina which are associated with a proliferation of connective tissue. They have also been observed after injuries to the optic nerve and retina. In most cases they lie on the papilla itself.

An apparent increase is seen when vessels that are otherwise invisible come into view as the result of distention, or of the fact that their walls have been made visible. Such an apparent increase is seen in commencing sclerosis of the vessels, in vascular engorgement from compression of the veins or glaucoma, in leucocythaemia, and in detachment of the retina. When the central artery is occluded the veins about the macula, which are otherwise scarcely visible, come plainly into view (Fig. **45**).

A surplus of vessels may be congenital, yet in most cases there is no oversupply, but the deceptive appearance is caused by the fact that the vessels which usually divide on the papilla, or after they have left it, have divided before they leave the hilus (see page **22**).

The presence of opticociliary, or retinociliary, vessels can scarcely be regarded as pathological; they are rather to be considered to be physiological variations (see page **23**).

The vessels which are to be observed in connection with other disturbances in the fundus, for example, with a coloboma of the chorioid, are rarely of a retinal, but usually of a chorioidal or scleral nature (Figs. **84** and **85**).

The *division* of the retinal vessels may present certain deviations from the normal. Thus it can be seen in many cases that some parts of the fundus usually provided with vessels are destitute of them, while other parts on the contrary show a superabundance. This condition may be either congenital, or be brought about by certain pathological processes, for example, by connective tissue formations which pull the retina in one direction or another. The subdivision may also deviate from the normal as the result of the destruction, or the new formation of vessels.

## D. The Course of the Individual Vessel

shows some typical peculiarities. The vessels in myopia, especially when it is of high degree, seem to be much stretched, or drawn out (Fig. **72**). On the contrary the course of the vessel is very crooked in hypermetropia, so much so that we sometimes speak of a tortuositas vasorum.

The disproportion between the size of the eyeball and the surface of the retina is the cause of this peculiar behavior, but in other cases the size of the caliber, i.e., the fullness of the vessel, is the actuating cause, so that a marked tortuosity is associated with a distention of a vessel, and a stretching, due to a simultaneous tension in its long axis, with its contraction.

The careful study of the course of the retinal vessels is of extremely great importance in the determination of differences of level within the eye. As we look into the eye of a patient with only one eye we cannot make use of our stereoscopic perception of depth, as this is dependent on binocular vision. We can perceive differences of level only by the aid of secondary means, among which the course of the vessels is a very efficient one. A number of fresh tubercles are shown along the course of the inferior temporal vein in Fig. **78**: the wavy course of the vessel enables us to perceive where the elevations are. In Fig. **47** is pictured a detachment of the retina: the folds of the detached retina may be recognized from the wavy courses of the retinal vessels. We judge in like manner concerning the conditions of level in a choked disk, in a commencing tumor, and in a proliferation of connective tissue (Fig. **23**).

How important the observation of the course of the vessels is in the diagnosis of glaucoma has already been pointed out on page 56.

## E. The Reflex

of the retinal vessels comes from those places that are vertical to the line of direction of the observing eye. It is strongest and broadest on the large vessels, yet it is visible, especially in young persons, on the minute branches, although it is weak and narrow: it is absent, however, even on vessels of medium size in senile eyes.

On the arteries it is generally brighter, more intense, narrower, and more

sharply defined than on the veins, yet the reverse condition is sometimes met with.

The reflex may undergo great individual fluctuations, under physiological conditions, with regard to intensity, uniformity, breadth, margins, color, and its comparative condition on the arteries and on the veins, while it cannot be determined in the individual case what has brought about the change. Still a few important points may be noticed.

The reflex is almost as useful as the wavy course of the vessels in the determination of differences of level, for at any elevation, no matter how slight, of the vessel above the level of the retina, the reflex disappears at the place where the bend takes place (Figs. **23, 24**).

The reflex from the vessels is totally absent in detachment of the retina (Fig. **46**). The slightest extravasation or œdema in the neighborhood of the vessel causes its reflex to disappear.

Under certain pathological conditions the color of the arteries approaches that of the veins, or the reverse, and then the reflexes will be similar to each other.

The more superficial the situation of the vessel, the more distinct and sharply defined is the reflex; the deeper the vessel, the less clear its light streak becomes.

When the blood pressure is decreased the reflex becomes broader, when increased it becomes narrower.

In the beginning of an arteriosclerosis the particularly intense, strikingly bright reflexes on certain parts of the vessel are the only signs of the disease. In the later course of the sclerosis, as atrophy sets in, they gradually disappear.

There is no agreement among authors as to the cause and place of origin of this reflex. Originally it was thought that the surface of the vessel itself reflected the light, but this idea has been abandoned, as the result of experiments and discussion. The theory that meets with the most favor is the one advanced by *Dimmer*, that the reflex comes from the surface of the column of blood in the veins, and from the place of the axial current in the arteries. Yet objections can be raised against this theory; *Elschnig*, for example, has shown that the reflex remains visible on the arteries after the circulation has ceased.

## F. Phenomena of Pulsation

*Venous and Arterial Pulse.*

Pulsation of the *retinal veins* is a normal phenomenon which is to be seen most distinctly in the large vessels at the hilus, at the place where they descend into the excavation. The veins best suited for observation are the large ones that end in a point or beak at the hilus. The blood column seems to be driven backward, toward the periphery at each stroke.

Two different pulsatory phenomena are included under the term *pulsation*

*of the retinal arteries:* 1, the compression pulse, which manifests itself in a greater and less fullness during the systole and the diastole; 2, rhythmic fluctuations of caliber.

The peculiar behavior of pulsation in the eye, that a venous pulse is normal and that an arterial pulse is not, is explained by the fact that the vessels are subjected to an external pressure, the so-called intraocular tension, while the vessels in other parts of the body are not. The pulsation of the arteries is prevented by this, and the veins, which have walls that afford little resistance, are compressed by it while they are less filled during the diastole. It is only when the relative conditions of pressure are changed, for example, when the *intraocular* tension is increased, or when the *intra-arterial* pressure rises abnormally high, or falls abnormally low, that an arterial pulse can be observed. The elevation of the intraocular tension can also be brought about by compression of the eyeball. If pressure is made with the finger upon the eyeball during an ophthalmoscopic examination, a venous pulse is seen at first and then, as the pressure is increased, an arterial pulse; finally, if the pressure is sufficiently forceful, the retinal vessels become completely empty of blood.

The *venous pulse* is met with frequently in normal eyes, but usually only in young people. In old age a distinct venous pulse is indicative of a commencing phlebosclerosis. Absence of the pulse when the eyeball is gently pressed by the finger is a symptom of thrombosis of the central vein.

An *arterial pulse* is always to be regarded as pathological. It indicates either an abnormally high blood pressure, an abnormally high pressure wave in the arterial system, as in cardiac hypertrophy, aortic insufficiency, aneurysm of the aorta or carotid, and exophthalmic goitre, or an abnormally low blood pressure, as after great loss of blood and in syncope, or, finally, an increased intraocular tension, as in glaucoma. Absence of the aortic pulse when the eyeball is pressed upon by the finger has been observed in occlusion of the central artery of the retina.

Retina

# Retina

## A. Preliminary Remarks on the Anatomy

In an eye that has been cut open the retina presents itself as a gray, cloudy membrane about $\frac{1}{3}$ mm thick, which separates easily from the pigment layer and is closely connected with the subjacent tissue in only two places,[1] the entrance of the optic nerve and the fovea centralis. When the retina is seized with forceps and removed from the eye the pigment layer remains in close connection with the chorioid. Nevertheless the layer of pigment epithelium belongs embryologically and physiologically to the retina, it is the outer layer of the secondary optic vesicle. The center of the retina appears yellowish, the macula lutea, with a dark brown point, the fovea centralis.

In the living, normal eye the retina is perfectly clear and transparent, so that its presence can be perceived only by means of the vessels that course in it.[2] The color of the fundus is therefore not influenced by that of the retina itself, but is due essentially to the greater or less abundance of the pigment contained in the pigment layer and in the chorioid; the color of the chorioidal vessels plays a subordinate part. The peculiar color of the macula is suppressed by that of the subjacent tissue; it appears only a little darker than its surroundings. Its center, the fovea centralis, is still darker, because the retina is very thin at that point and consequently the chorioid and pigment epithelium show through with special clearness. The contrast with its surroundings becomes particularly marked when the retina in the vicinity of the macula is cloudy, as in occlusion of the central artery (see Fig. **44**).

The layers of the retina are known as the **outer and the inner,** according to their positions relative to the contents of the eye. Those lying nearest to the vitreous are called the inner layers, those more distant the outer. The outer layers, i.e., the layer of rods and cones, the outer granular layer, and the membrana limitans, which lies between them, form what is known as the layer of *sensory epithelium*, while the others, which occupy the inner portion of the retina, are grouped together as the cerebral layer.

This is a division which is not simply anatomical, but is of great clinical and diagnostic importance, as the two layers receive nutrition from different sets of vessels.

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[1] The retina is also attached to the chorioid at the ora serrata.—*F.*

[2] A delicate striation, radiating from the papilla, is visible in many normal eyes; this is to be referred to the color of the retina itself.

A full and precise description of the anatomy would require too much space, so only those points will be mentioned which are of considerable clinical importance. The layers of nerve fibers consist of bundles of naked nerve fibers which lace, as it were, into a plexus. It is only in exceptional cases that they have medullary sheaths and form a white spot (medullated nerve fibers, see Fig. 9). The fibers radiate from the papilla, with the exception of those



Layer of pigment epithelium
Layer of rods and cones
Membrana limitans externa
Outer granular layer
Outer molecular layer
Inner granular layer
Inner molecular layer
Layer of ganglion cells
Layer of optic nerve fibers

Layer of pigment epithelium
Layer of rods and cones
Layer of granules and visual cells
Outer plexiform layer
Layer of horizontal cells
Layer of bipolar cells
Layer of amacrine cells
Inner plexiform layer
Layer of ganglion cells
Layer of optic nerve fibers

Fig. Q.

Anatomy of the Retina, after *Greeff*.

coming from its temporal side, which circle in a great arch about the macula. The macula itself is supplied by particularly fine fibers which run directly to it from the temporal margin of the papilla (the papillomacular bundle, see pages 16 and 58). The construction of the retina undergoes changes both at the macula and at the periphery, which are of clinical interest.

The **macula** is situated about $1\frac{1}{2}$ papillary diameters (4 mm) outward and a little downward from the entrance of the optic nerve, in the inverted image of course inward and upward, is usually transversely oval, less often round, and measures in its horizontal diameter 1.7 to 2 mm. Its margins are a little raised, and in its center is a depression with level margins, the fovea centralis. The number of the cones increases toward the fovea at the expense of the rods.

To give a very rough idea of the anatomy it may be imagined that the cone fibers, and with them their connections, are combed apart, or parted like hair, at the fovea, so as to lay bare the cones at the bottom of the part.

The **peripheral portions.** While the number of the cones increases as the macula is approached, it decreases toward the periphery, where the rods preponderate in number considerably. Both cease at the ora serrata. The other layers also blend at this place until only a single layer of cylin-

drical cells is left, beneath which lies the pigment layer, with which it forms an intimate connection as it passes over to the iris.

The **nutrition of the retina** is derived from two sources: 1, the central artery, 2, the vessels of the chorioid. The former supplies the cerebral layer, the latter the layer of nerve and pigment epithelium.

1. The principal branches of the *central artery of the retina* run close to the inner surface of the layer of nerve fibers, or project above this into the vitreous, but are always covered by a few bundles of nerve fibers.

2. The layer of nerve epithelium is entirely without blood vessels and receives its nourishment by diffusion from the capillary network of the chorioid; the fovea centralis is likewise nonvascular, the fine retinal vessels end in a circle of capillary loops on its margin.

**Physiology.** The percipient organs of the retina are the rods and cones, but these lie in its outer layer, toward the sclera. This gives the peculiar condition that the rays of light must first pass through the entire thickness of the retina in order to reach the organs of perception. Hence the necessity of the perfect transparency of the retina, and of the absence of vessels in the region of the macula.

## B.  General Diagnosis

*Ophthalmoscopic Differentiation of Diseases of the Inner and Outer Layers of the Retina and of the Chorioid*

As the pigment epithelium as well as the neuroepithelium receives its nutrition from the choriocapillaris of the chorioid, the appearance of pigment changes is the characteristic symptom of the ophthalmoscopic picture produced by a disturbance in this vascular region. On account of the simultaneous involvement of the retina and the chorioid we do not speak in these cases of a retinitis, but of a chorioretinitis. Although in rare cases a pigmentation of the retina may take place as the result of a retinal hemorrhage, yet in the majority the following statement is correct:

*The appearance of pigment stains, and of abnormal accumulations of pigment, in the retina indicates a disease of its outer layers, or of the chorioid, a chorioretinitis; diseases of the retina without involvement of the pigment are, on the contrary, of its inner layers.*

*The Position of the Changes in the Retina,*

i.e., the depth at which they lie, can be determined ophthalmoscopically:

1. From their relations to the retinal vessels. If the latter pass over the former the changes are deeper than the vessels and therefore are in the deeper layers of the retina (Fig. **31**). If, on the contrary, the vessels are partly or wholly covered, the changes lie in the superficial layers (Fig. **33**). Should the vessels be completely hidden, as by a hemorrhage, the change must be upon the retina, and the hemorrhage, in the example cited, is called preretinal.

2. From their form and arrangement. Striated patches or hemorrhages (Fig. **32**), especially such as extend in the form of rays from the papilla, or accompany the larger vessels (see Fig. **28**), lie in the most superficial layers and follow in them the course of the nerve fibers. When they are irregular, or inclined to be round, they usually lie in the deeper layers (Fig. **35**). An exception to this rule is formed by the stellate patches in the macula seen in albuminuric retinitis (Fig. **34**), which lie, in spite of their radiation, in the deeper layers of the retina.

3. From the presence or absence of anomalies of pigment. The presence of these shows that the changes lie, in part at least, in the deepest layers of the retina.

*Retinitis.*

The name retinitis is used to indicate things with which inflammation has nothing to do. A reform of ophthalmological nomenclature is greatly needed here. As soon as hemorrhages, or bright spots, or dark spots are visible in the fundus, the patient is said to have retinitis, no matter whether they are foci of inflammation, products of degeneration, or the results produced by altered vessels. To be sure, inflammation and degeneration can scarcely be discriminated in the ophthalmoscopic picture, and the pathological conditions that have been found in other similar cases must be taken into account in order to be able to determine the disease in any particular case. Usually that which is called retinitis, and is manifested in the form of diffuse, or circumscribed bright spots, or of hemorrhages, is the result of diseases of the vessels.

The extremely sensitive tissue of the retina, with its very small capillaries, reacts with great ease to any disturbance of circulation, and likewise any change in the composition of the blood or tissue juice leaves its trace in the retina. Often the very first signs of a general disease are made visible in the retina because it is so very sensitive.

Unfortunately the manifestations in the eye of the various constitutional diseases are remarkably alike, so that it is only in rare cases that the exact etiological diagnosis can be made from the ophthalmoscopic picture alone, it usually has to be learned from the results of a general examination. Even though it is not always possible to make the **etiological diagnosis** from the ophthalmoscopic examination, yet this much can be learned, *that a general disease is present in all cases in which fresh changes are found in the form of white or black spots, opacities, or hemorrhages. In such a case it is our imperative duty to submit the body to a very thorough examination, paying particular attention to the urine.*

## Are Alterations in the Pigment Epithelium Present or Not?

The inner layers of the retina are nourished by the central artery, while the outer, together with the pigment epithelium, derive their nutriment from

the chorioid, as has been already pointed out. Disturbances in the central artery of the retina therefore are made manifest by changes in the inner layers, those in the chorioid by changes in the outer layers and particularly in the pigment epithelium. This is the reason why the question, whether alterations are present or not in the pigment epithelium, is of such great importance, for the answer to this question is decisive with regard to the seat of the disease and the vascular system that is affected.

The alteration of the pigment epithelium may manifest itself in two ways, either as a depigmentation, i.e., an atrophy of the pigment layer, or as an abnormal accumulation of pigment. Depigmentation lays bare the tissue of the chorioid and allows the chorioidal vessels, which are more or less changed in such cases, to be seen, when the sclera itself is not laid bare by the simultaneous atrophy of the chorioid. This gives rise to white spots, which need to be differentiated from the white spots of the retina. For the differential diagnosis see page 125.

## C. Special Diagnosis

We have made quite a digression into a rather theoretical field and will now return to the practical diagnosis.

We have studied the papilla and its vicinity with the greatest care, we have observed the retinal vessels from the various points of view, and now we turn to the diagnosis of the special diseases of the retina. We will be guided first by the question,

*Are Pigment Changes Present or Not?*

and will deal first with

## Retinal Lesions Which Exhibit No Alterations in the Pigment Epithelium

(Diseases of the inner layers.)

These are mainly

Hemorrhages,
White Spots, and
Diffuse Opacities.

These will guide us further. Naturally all three disturbances may be present at the same time. Hemorrhages and white spots occur together very often. So, too, are diseases of the optic nerve often combined with these changes in the retina. Hence we will make the following subdivisions in order to proceed in the differential diagnosis:

*I. Hemorrhages:*

*(a) As the only, or the most important change in the retina, perhaps in combination with changes in the retinal vessels;*

*(b) in connection with diseases of the optic nerve and its vicinity.*

*II. White spots with or without hemorrhages or diseases of the optic nerve.*

*III. Diffuse opacities.*

# I. HEMORRHAGES

## (a) Hemorrhages as the Only, or the Most Important Change in the Retina.

After what has been said it may seem unnecessary to call attention to the significance of hemorrhages in the fundus, but this definite statement may be repeated on account of the importance of the subject: *When injuries, high myopia, and glaucoma can be excluded hemorrhages are always signs of a general disease.* They form a signal of warning; there is something out of order in the organism; make a general examination. Even when the hemorrhage is as slight as that to be seen in Fig. **20**, it may and must be made the starting-point of the diagnosis of a serious disease.

After hemorrhages have been found *special questions arise concerning their size, abundance, and position.*

Hemorrhages may be of the most varied extent; sometimes we find little, circumscribed patches, which can be seen only after a very careful search with the pupil dilated, and in the upright image alone. The only advice that can be given the physician—and this implies no question as to his skill—is to dilate the pupils in a doubtful case in order to be able to make a more accurate examination. The only precautions to be observed are those given on page 11.

On the other hand, the hemorrhages may be so massive as to form the most prominent feature of the ophthalmoscopic picture, as to cover every detail, and to make the fundus look like a single lake of blood (see Fig. **27**). The number also of the hemorrhages may vary extremely.

As regards the depth at which they are situated it has already been said that fine, striated hemorrhages which radiate from the papilla lie quite superficially in the layer of nerve fibers, while roundish, or lumpy ones are to be sought in the middle or deep layers. Hemorrhages that cover the retinal vessels lie in front of the retina and are called preretinal; they are usually round, or oval, in form, and are found for the most part in the region of the macula.

Topographically the following points have to be noted: the position of the hemorrhages relative to the papilla, to the macula, and to the large vessels, whether they are diffusely distributed, and whether they are in the vitreous.

Small hemorrhages come from the capillaries, large ones from the larger vessels. It is often impossible to differentiate between a venous and an arterial hemorrhage, and the differentiation is of little use. When differences in color are to be seen they are to be ascribed to the different ages of the hemorrhages, as fresh ones are of a bright, blood red, while old ones are of a dark, brown red.

NOTE. The absorption of hemorrhages takes place pretty slowly as a rule. The spots gradually become darker, until they finally disappear without leaving any traces, or they become transformed into white spots, which in turn become invisible; it is only in rare cases that masses of connective tissue and pigment remain as traces of a hemorrhage. On the other hand, hemorrhages may last for months. The impairment of the functions of the retina depends on the situation, the force exerted at the time when they occurred, and their size. An attack of glaucoma may be mentioned as one of the possible consequences of a severe intraocular hemorrhage, but, inversely, glaucoma may also be the cause of hemorrhage (see page 120).

## Is a Differential Diagnosis Possible, Based on These Findings?

It is in many cases:

1. *From the extent.* If we find as extensive a hemorrhage as that shown in Fig. **27** we can confidently base upon it the diagnosis of **thrombosis of the main trunk of the central vein.** This clinical picture was named by *v. Michel* apoplexia sanguinea and compared by him with corresponding hemorrhages in the brain. When we study the details of the picture we are struck by the fact that we cannot actually see the vessels, except above and very close to the papilla. The veins are dark red, almost black, and tortuous in places, while the only visible artery is small and exhibits a strikingly distinct reflex light streak, which indicates a commencing sclerosis of the vessel wall. The papilla is still fairly visible in this case, but frequently it is involved in the area covered by the hemorrhage, when its margins are totally hidden. The thrombosis is a consequence of arteriosclerosis, or perhaps of syphilis, or of nephritis, and therefore its prognostic signification is grave (see page 153).

2. *From the position.* If the hemorrhages are not spread all over the fundus, but only occur *along one or more vessels*, as shown in Fig. **28**, we may speak of a **partial thrombosis,** especially when the vessel itself shows alterations. The vascular changes could not be studied in the last picture on account of the enormous number of hemorrhages, but in this one they can be studied very well. The thrombosed vein is broadened on the whole, its caliber varies in different places, it is accompanied by abnormal reflexes, and is filled by a very dark, almost black, column of blood. The cause is arteriosclerosis, syphilis, or nephritis.

*Isolated hemorrhages* in the macula are usually of an arteriosclerotic nature when a high degree of myopia is not present, and an injury can be excluded.

3. *From the form.* Little shuttlelike hemorrhages, each with a white spot in its center, are commonly caused by such diseases of the blood as anæmia and leucocythæmia.

No definite etiological conclusion can be drawn in any other case from the condition of the hemorrhage. In all other cases the result of the general examination must be awaited. Such an examination must be made in those cases that have been mentioned as well, because the arteriosclerosis that may exist may be complicated by other diseases. The causes of a retinal hemorrhage are very many.

## The Causes of a Retinal Hemorrhage

The first cause to be mentioned is an injury. It is by no means necessary that the eyeball itself shall be wounded, hemorrhages result from contusions, and are particularly common when the eye has been struck by a ball. In these latter cases special attention should be paid to the macula in the examination.

They are also found after severe injuries of the body, such as compressions of the thorax. They may appear congenitally as the result of traumatism during labor. More rarely they are to be seen as the result of the penetration of a foreign body into the eye, as shown in Fig. **29**. Emphasis is to be laid on the word "seen" in this connection, as they are often present though invisible because of the hemorrhage that takes place into the vitreous at the same time.

Their most important cause is **arteriosclerosis,** and in these cases they are of very great prognostic value because, in at least 50%, they are forerunners of hemorrhage into the brain (see page 153). These hemorrhages have been studied already under the forms of total and partial *thrombosis* of the central vein, and of the isolated *hemorrhage* in the *macula*, but they are also found distributed about in the retina as *diffuse spots*, which give a very bad prognosis with regard to the later onset of apoplexy, from 80 to 100%, while a thrombosis is followed by apoplexy in only about 50%. Finally, it may be mentioned, for the sake of completeness, that arteriosclerosis may manifest itself through a hemorrhage into the *vitreous.*

Reference is to be made to the chapter on "The Changes in the Vessels of the Retina," and the very careful study of these vessels is urged upon the reader.

**Diabetes** and **nephritis,** especially the form of the latter characterized by the granular atrophy of the kidney, are very important causes both of little, stippled hemorrhages, and of large, lakelike ones; both forms usually appear at the same time with white spots, or disease of the optic nerve, so

the description of them is reserved for a later chapter. This etiology is always to be borne in mind when the hemorrhages are isolated.

Syphilis is likewise one of the principal causes of retinal hemorrhages, but other manifestations of the disease are usually present.

All of the remaining causes are much less frequent. First among them come the **diseases of the blood.** It has already been said that these hemorrhages often present a special form, a spindle, or shuttle shape with a white spot in the center, yet they may not have this peculiarity, but may appear simply as small spots and stripes. Hemorrhages are rarely met with in chlorosis, they are more common in pernicious anæmia, and occur most often in leucocythæmia, chiefly in the form of striæ. In well marked cases of the last mentioned disease the orange tone of the fundus and the great breadth of the vessels are diagnostic.

Similar hemorrhages are also observed in simple anæmia following a great loss of blood, and in hæmophilia.

It hardly needs to be said that they occur in the **hemorrhagic diathesis,** as well as in purpura, scurvy, purpura hæmorrhagica, and *Barlow's* disease, but they are of comparatively little importance in these diseases, as they are all rare.

The appearance of hemorrhages is of considerable importance, on the contrary, in the acute **infectious diseases,** such as malaria, typhoid fever, influenza, miliary tuberculosis, and sepsis, though they are not so very frequent in them. Sometimes they are to be seen in cancerous cachexia.

Conditions that need to be differentiated from retinal hemorrhages are:

1. *Lacerations* in an opaque, detached retina. These lie at a deeper level than their surroundings, as can be proved by parallactic displacement, have a distinctly opaque, gray margin, and sometimes allow the markings of the chorioid to be seen through them.

2. *Hemorrhages* in the chorioid. These are rare and demonstrable only when the fundus contains little pigment. The vessels of the retina, and perhaps of the chorioid, pass over them. Other changes are generally present in the chorioid. They are of practical importance only in myopia and eclampsia.

3. The *cherry red spot* in occlusion of the central artery (see page 150). The demonstration of an acute, diffuse cloudiness of the retina in these cases makes the diagnosis certain.

4. *Spots of pigment.* These are dark and usually are found in company with changes in the chorioid.

5. The traumatic perforation of the macula. This is rare and is characterized by a circular blood red disk in the macula, about two thirds the size of the papilla, that looks as if cut out with a punch.

As the so-called **recurrent hemorrhage into the vitreous** is looked upon by many authors as a hemorrhage from the retinal vessels, it should be mentioned in this place. This is met with in young persons, usually males.

and has the unpleasant tendency to recur that is implied by the name. The real cause is unknown, though it has been supposed to be due to an early, localized arteriosclerosis.

## (b) Hemorrhage into the Retina as an Accompanying Symptom of Disease of the Optic Nerve (without white or black spots)

*Glaucomatous Excavation.*

As soon as the media have cleared up sufficiently, after an acute attack of glaucoma, to allow the details of the fundus to become visible again, hemorrhages, varying in form and number, can be seen in addition to the excavated papilla and the more or less altered blood vessels. Usually they form little linear, or blotlike spots, which radiate from the papilla far out into the periphery. The prognosis is generally bad in such a case of glaucoma.

In this connection may be mentioned the hemorrhages that take place after, or during operations on glaucomatous eyes as the result upon the vessels of the sudden fall of the intraocular tension.

*Optic Neuritis.*

Hemorrhages are found near the papilla in optic neuritis from all manner of causes, but they are particularly common in that due to nephritis and diabetes. The great diagnostic importance of these hemorrhages was pointed out when speaking of the so-called pseudoneuritis. The presence of a single minute hemorrhage in a doubtful case of pseudoneuritis or optic neuritis immediately renders the latter diagnosis positive.

*Choked Disk.*

Fine, striated hemorrhages, which, from their form, must lie in the layer of nerve fibers, form an almost regular feature of choked disk.

## PLATE XV

Fig. 27.   Occlusion of the Central Vein of the Retina (Apoplexia Sanguinea Retinae)

Fig. 28.   Occlusion, or Thrombosis, of a Single Vein of the Retina

# Fig. 27.—Occlusion of the Central Vein of the Retina (Apoplexia Sanguinea Retinae)

The striated arrangement of the hemorrhages shows that they are situated in the most superficial layers of the retina. The portions of the retina that lie between the hemorrhages are slightly œdematous (see page 117).

The vessels are, for the most part, concealed by the hemorrhages, and are also in part thrombosed, so that only those in the portions closely adjacent to the papilla are visible.

The papilla itself is not materially changed, but it may be covered by the hemorrhages.

The cause of such an occlusion is nephritis, or arteriosclerosis in most cases.

# Fig. 28.—Occlusion, or Thrombosis, of a Single Vein of the Retina

The changes in the vessels, which could not be seen in the preceding picture because of the profuse hemorrhages, can be perceived quite well in this one.

The thrombosed vein is very broad, is of a deep, dark red, fluctuates a great deal in caliber, has abnormal reflexes and has a marked tortuosity.

The hemorrhages are in the immediate vicinity of the diseased vessel and thereby betray the cause of the change.

The arteries are normal in these cases, yet the afferent branch is frequently small (see page 117).

Tab. **15.**
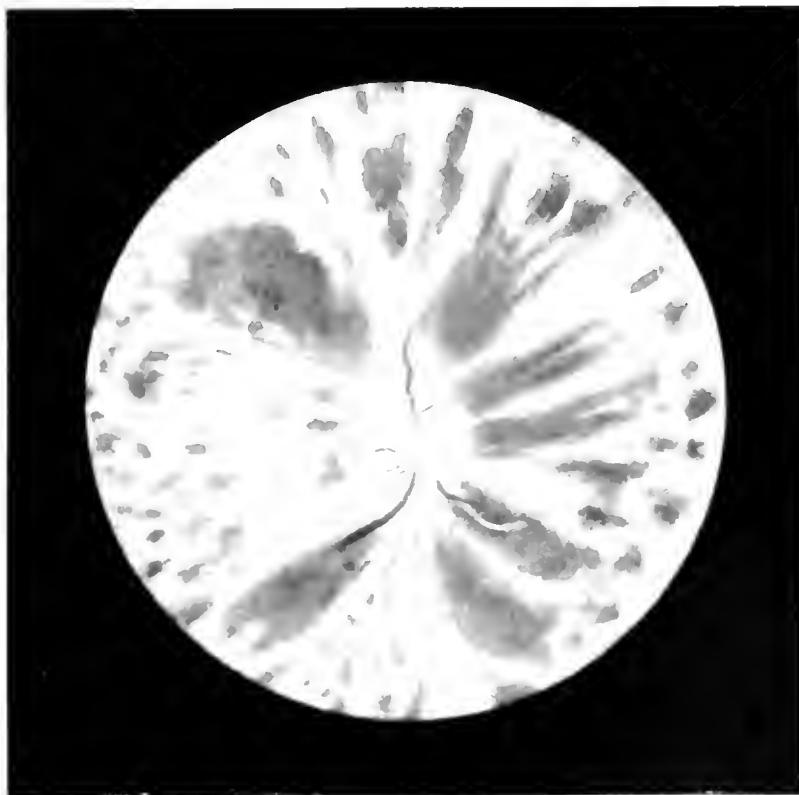


Fig. 27.
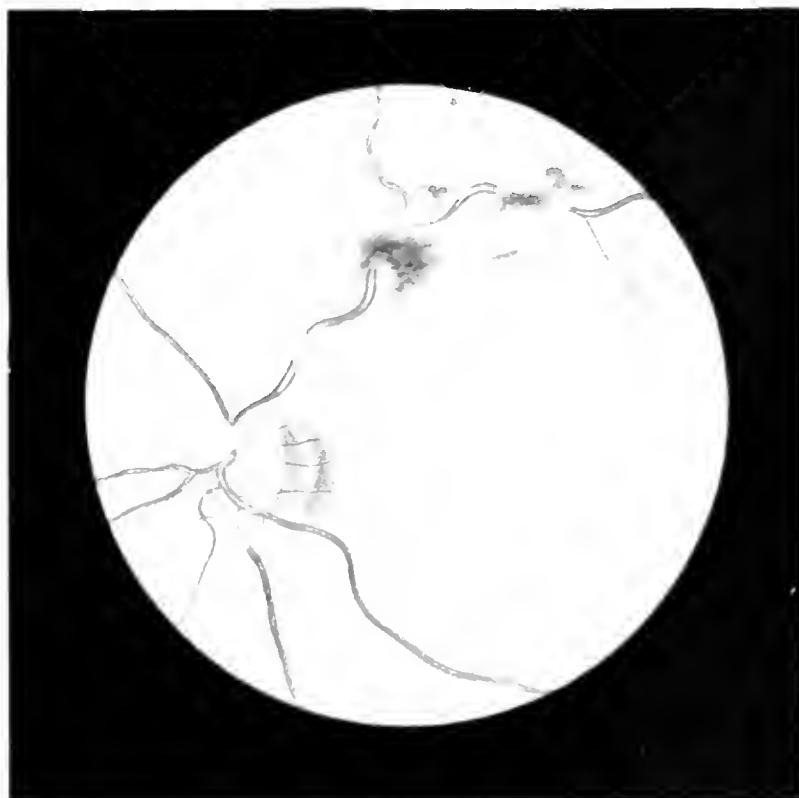


Fig. 28.

PLATE XVI

Fig. 29.   Foreign Body in the Retina and Chorioid

## Fig. 29.– Foreign Body in the Retina and Chorioid

The little, gray, silvery bit of steel did not have force enough to perforate the eyeball a second time; it could only tear its way into the retina and chorioid, where it remains.

The torn place is partially covered by a hemorrhage and is surrounded by an œdematous, gray oval. This piece of steel was removed by means of a magnet and good vision was preserved, but the latter may be badly impaired by the onset of a detachment of the retina. The picture is usually very indistinct, because of an accompanying hemorrhage into the vitreons; if this latter is absent it is generally possible to obtain a glimpse of the foreign body.

Tab. **16.**
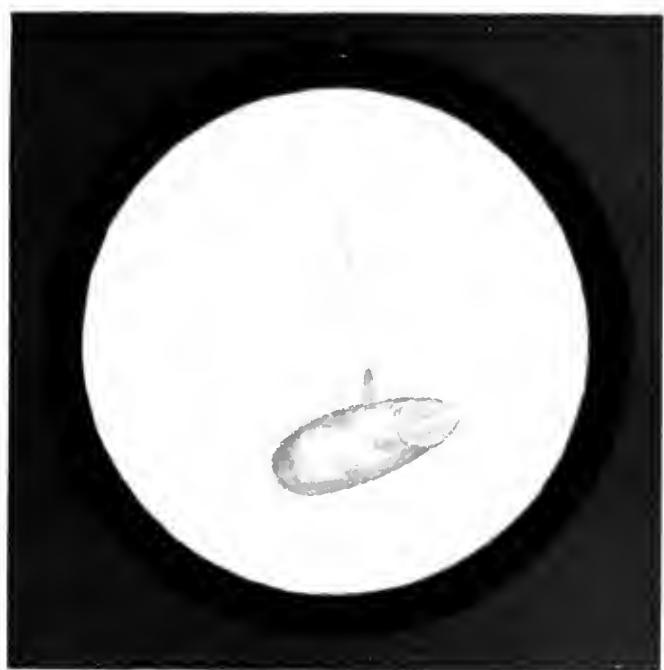


Fig. 29.

## II. WHITE SPOTS IN THE FUNDUS

### Question 1

### *Is the White Spot in the Retina, or in the Chorioid?*

(*Differential Diagnosis between Retinal and Chorioidal Spots*)

Unfortunately there is no quite exact rule by which it can be determined positively in every case whether a certain white spot in the fundus is in the retina or in the chorioid. The reason why is apparent when we consider that in many cases, as in the case of miliary tuberculosis pictured in Fig. **81,** we do not see the chorioidal affection itself, but have to be satisfied with the observation of the secondary symptoms which are produced by a lesion in the chorioid upon the retina that lies over it, for example, of a circumscribed œdema. Still, in spite of the fact that we can see only the secondary symptoms in the retina, we can make the diagnosis of tubercle of the chorioid in this case, because our clinical and pathological experience is that under certain conditions of high fever and stupor the appearance of circumscribed patches of retinal œdema in the fundus are indicative of tubercle of the chorioid. We must be guided many times in this way by our general clinical and pathological experience in deciding whether the lesion is in the retina, or in the chorioid, but this may fail if we do not use at the same time our special ophthalmological knowledge. Spots may appear in sepsis, and therefore be attended by quite similar general symptoms, which may seem very like those of miliary tuberculosis, but they are situated in the retina, as has been proven by pathological examination. Hence the general symptoms may guide us wrongly in these cases if we do not know that hemorrhages occur far more often in septic retinitis than in miliary tuberculosis, while on the other hand, a simultaneous involvement of the optic nerve points rather to the latter.

I do not wish to make a careful observer timid, or to stump a young ophthalmologist with such an unsatisfactory case, but only to show him by this example that the answer to the question *whether the lesion is in the chorioid or in the retina, is not to be learned through the* **vision** *alone; it is to be obtained only from the most careful consideration of the other conditions in the eye and the utilization of general clinical and pathological knowledge.*

A number of symptoms can always be found which will make the diagnosis certain in one way or another in any given case. When we see, for example, in Fig. **38,** that the white spot lies partially over a vessel in the retina, we know positively that the lesion is retinal, and when, on the other hand, we see in Fig. **74** that the patch is surrounded by a beautiful wreath of pigment, we need nothing more to prove that the disease is of the chorioid. Unfortunately these positive signs are not always present; in Fig. **31** the vessels

of the retina run over the white spots, and in Fig. **81** the white spots have no pigmented borders, yet in the one the lesion is in the retina, and in the other in the chorioid. Therefore we have to differentiate between those symptoms that are trustworthy, or nearly so, and those that are adjuvant.

*Differentiation between Lesions in the Chorioid and the Retina.*

1. *Trustworthy symptoms.* These can be accepted only in a positive sense, i.e., their absence does not prove the contrary.

| Retinal Lesion | Chorioidal Lesion |
|---|---|
| 1. The partial covering of a retinal vessel by a white spot (Fig. **38**). | 1. Pigment deposited about or on the spot (Fig. **71**). |
| 2. A stellate form in the macula (Fig. **34**). | 2. The demonstration of chorioidal vessels, whether sclerosed or not, in the spot (Fig. **64**). |
| 3. The demonstration of hemorrhages in the retina, and of changes in the retinal vessels is relatively certain (Fig. **35**). | 3. A markedly crescentic form, as in Fig. **83**. |

N. B.—If both varieties of spots are found in any case, changes are probably present in both membranes; thus, in Fig. **50,** the retinal vessels are partly covered by the spot, and yet there are heaps of pigment at its upper margin. In this case there was a fresh disease of the retina, glioma, and an older disease of the chorioid, perhaps due to hereditary syphilis.

2. *Adjuvant Symptoms:*

| | Retinal Lesion | Chorioidal Lesion |
|---|---|---|
| Color: | Bright, often brilliant white, more rarely yellowish or reddish. Œdematous spots gray. | Fresh: yellowish, reddish yellow, slightly gray. Old: white, but with pigment. |
| Size: | Usually small, or formed by the confluence of smaller spots. | Variable. A large white patch below (in the inverted image above) the optic nerve indicates a coloboma of the chorioid. |
| Position: | Very rarely in the periphery. | Often in the periphery. |
| Visibility of the vessels of the chorioid: | In the case of a tesselated or albinotic fundus the vessels of the chorioid are usually invisible in the vicinity of the change in the retina because of the opacity of the elsewhere transparent membrane (Fig. **22**). | In the case of a uniform, stippled fundus the vessels of the chorioid stand forth and indicate early changes in that membrane. Notice the partially albinotic fundus in Fig. **57**. |

In spite of all this help there remains quite a number of cases in which the diagnosis may be doubtful, and it is necessary that we should be acquainted with these in order to avoid falling into error.

(a) *Changes in the Retina.*

1. The *coronula*, in a case in which a total *occlusion of the central artery* took place at some former time. A number of fine bright, yellow or reddish spots are to be seen arranged in a circle in the macula, and are usually surrounded by a slight pigmentation (Fig. **16**). The correct diagnosis is indicated by the atrophy of the optic nerve and the extreme smallness of the retinal arteries.

(b) *Changes in the Chorioid.*

1. Aside from this circular arrangement, *arteriosclerotic changes in the macula* (Fig. **62**) may form an ophthalmoscopic picture which is quite similar to that presented by the coronula. The results of the general examination, the other arteriosclerotic changes in the eye, and the age, must all be taken into account in making the diagnosis. These changes are due to a circumscribed hyaline degeneration of the choriocapillaris.

2. *Colloid Formations on the Vitreous Lamella* (Fig. **41**). These appear as little, bright points, of a yellowish, or yellowish gray color, which can be differentiated from the typical spots in the retina by their color and by the indistinctness of their outlines. Pathologically, they are small, knoblike thickenings of the vitreous lamella of the chorioid, which mechanically destroy the pigment epithelium at the places where they exist. But, as this leads in turn to accumulations of pigment, little black lumps can generally be seen in their vicinity.

3. *Little Foci in Sympathetic Inflammation* (Fig. **40**). Little sharply defined spots, whitish, yellowish white, or reddish yellow in color, are to be seen sometimes in the periphery of the fundus of an eye that is sympathetically inflamed. They are usually round, more rarely oval, and have little tendency to blend. Sometimes there is a brownish tesselation in their vicinity, although a true pigmented edge, or a marked accumulation of pigment, is absent. For this reason, which indicates a retinal affection, some authors believe these spots to belong in fact to the retina, but we must look upon them as appertaining to the chorioid because of the nature of the disease as a whole, and because of the anomalies of pigment occasionally to be observed. When other symptoms of sympathetic inflammation are present at the same time the diagnosis cannot fail to be made.

N. B.—Attention must be called to a possibility of error that arises from mistaking reflections from the retina for pathological changes. These may appear just like little yellowish patches in the macula, but they disappear when the pupil is dilated and the examination is made by the direct method. They also change their forms when the mirror is rotated.

*After the diagnosis of a disease of the retina has been established in the above manner there arises*

## *Is This a Case of Medullated Nerve Fibers or Not?*

Medullated nerve fibers occupy a unique position among the affections of the retina, so it seems best to deal with them separately. The entire nature of the condition is expressed by the term medullated nerve fibers, while all other white spots in the retina are only symptomatic of causes that must be ascertained through other conditions.

*Diagnosis and Importance of*
*Medullated Nerve Fibers.*

Medullated nerve fibers (Fig. **9**) are quite superficial, in harmony with their anatomical development, and thus partly cover the vessels of the retina. They radiate from the papilla and show a more or less distinct fibrillation, corresponding to the course and the construction of the fibers, which is particularly evident at the margins of the patches that they form, where they often terminate in a flamelike figure. They are for the most part slightly yellowish in color, and commonly are in immediate connection with the papilla, though occasionally separated from it, when they follow the course of the large vessels. The diagnosis is somewhat more difficult in the latter cases, but the difficulty is removed by observation of the markings of the fibers and, above all, by the absence of any other lesion. The great stress that is to be laid on the latter condition is shown by the lesion pictured in Fig. **32**, which agrees throughout with the description given of medullated nerve fibers, although it rarely happens that such fibers overlap the entire margin of the papilla, but is proved not to be such by the little hemorrhages at the margin of the lesion.

Medullated nerve fibers form a congenital anomaly and are therefore of no other clinical importance.

After these have been excluded comes

## *Of What Nature Are the Spots in the Retina?*

This question must be divided into two: 1, with regard to the pathology; 2, with regard to the etiology of the spots. The spots may differ greatly in their pathological construction, and yet arise from the same causes, or, on the other hand, they may be due to quite different causes and present the same pathological picture.

# In how far can the Pathological Construction of a Spot be Determined from the Ophthalmoscopic Picture?

*Differential Diagnosis of White Spots from a Pathological Standpoint*

Leaving medullated nerve fibers out of consideration, white spots may be due to connective tissue, to proliferation of the glia, to varicose thickening of the layer of nerve fibers, to fatty degeneration, to œdema, to fibrinous or serous exudates, to deposits of calcareous matter, or to hyaline degeneration.

1. *Connective Tissue* (Figs. **36** and **37**).—The presence of connective tissue always indicates that a serious disturbance has taken place in the retina, except in the rare cases in which it is present congenitally. In the majority of cases it starts from the vessels, or from their adventitial sheaths, and consequently is almost always seen in connection with vessels that are either pathologically changed or newly formed. These are cases of retinitis proliferans.

The development of connective tissue is caused by hemorrhages, or, in a small number of cases, by lacerations of the chorioid and retina. These naturally present no typical arrangement (Fig. **82**).

The color is like that of medullated nerve fibers, except that it is rather duller, and they often have a similar striated structure. Usually it can be determined by parallactic displacement that they project above the level of the retina. Aside from the things already mentioned, arteriosclerosis plays the most important part in their etiology, then comes syphilis, and, in the third place, diabetes. In the latter disease the masses of connective tissue are particularly well marked and project far into the vitreous, as in Fig. **36**.

2. *Œdema.*—Only those cases in which the œdema is circumscribed are included here; those in which it is diffuse, as it usually is, do not enter into the question (for these see page 149).

The diagnosis is particularly difficult when œdema occurs at the same time with other affections of the retina, but its presence may be suspected in every serious retinal disease.

The diagnosis is easy only when it occurs in isolated patches. It is then usually found over fresh chorioretinitic lesions, especially tubercle nodules, when it manifests itself in the form of medium-sized, roundish spots of a light gray color, with obliterated margins and a distinct elevation, which can be determined particularly well when retinal vessels pass over it (Fig. **78**). Although only the œdema of the retina is to be seen in such cases we are accustomed to make from it the diagnosis of a lesion in the chorioid.

3. The *varicose thickening of the layer of nerve fibers* has the same substratum as the medullated nerve fibers and often resembles the latter very

closely. The altered fibers are of a light, golden white, have a certain bril
liancy in places, and radiate in striae from the papilla. This change is met
with in the greatest variety of diseases, as a local phenomenon of neuro-
retinitis of albuminuric and other origin, in choked disk, in diseases of the
vessels, and so on. It often covers or envelops the vessels of the retina as
it is quite superficial in its situation (Fig. **32**).

4. *Fatty Degeneration.*—This is certainly the principal cause of the white
spots. To it are to be ascribed the white spots ordinarily to be seen in
albuminuric retinitis. They appear ophthalmoscopically as yellowish, or pure
white (Figs. **30** and **31**), small, roundish patches which often blend, and so
produce large spots. They lie mainly in the intergranular layer and have
a special predilection for *Mueller's* supporting fibers. The vessels of the
retina may be seen to glide over these patches. The granules of fat are also
to be found in the layer of nerve fibers, the layer of ganglion cells, and the
internal granular layer, but to a much less degree; they are never present
in the layer of visual cells, or in the outer granular layer, which have a
different nutritive supply (see page 113).

The stellate figure in the macula is brought about by the fatty degenera-
tion of the supporting fibers, or, according to others, of *Henle's* layer.

This fatty degeneration is met with in all manner of diseases, but chiefly
in nephritis and diabetes.

5. *Fibrinous exudates* may lie in any of the inner layers of the retina
and consequently may be in various relations to the blood vessels. The spots
thus produced are larger than those caused by fatty degeneration, and some-
times they induce a slight elevation of the part affected (Figs. **38** and **39**).
When they are superficial they are of a light blue white color and look as
if they were loosened up, like bits of cotton.

N. B.— Sometimes little glittering points or needles can be seen in the
retina. These are crystals of cholesterin, such as are to be seen sometimes
floating in the vitreous. When the latter are situated in the most posterior
layers of the vitreous they may perhaps simulate spots in the retina, but their
great parallactic displacement, and the fact that they can be seen when we
simply throw light into the eye, prove them to be what they are, the so-called
synchisis scintillans.

None of the other pathological changes mentioned above can be recog-
nized with certainty from the ophthalmoscopic picture.

*The Course*

of the changes that have been mentioned is usually very slow. In mild
cases the symptoms undergo involution after some weeks and the eye may
return to its normal condition, both functionally and ophthalmoscopically.
The little white spots caused by fatty degeneration gradually become redder,
ill defined, and then blend with their surroundings. Fresh spots are therefore
bright white and sharply defined, while older ones are reddish and ill defined.

A prolonged duration of the disease finally injures the nervous elements more or less, so that at least an inpairment of the functions remains, and in many cases changes are left that can be seen with the ophthalmoscope, such as a migration of pigment into the atrophic retina, changes in the vessels, and atrophy of the optic nerve.

In other cases atrophy of the papilla results from atrophy of the nerve fibers. A case is pictured in Fig. **16** in which a destruction of the nerve fibers, and, as a result, an atrophy of the optic papilla, was caused by an occlusion of the central artery. Pigment cells have migrated into the atrophic retina around the papilla and in the region of the macula. In other cases the change may be located very near the papilla, as in Figs. **32** and **33**, when the consequently atrophied disk will probably show very indistinct margins.

In still other cases such a picture may finally be produced by the increase of the changes, as that shown in Fig. **34**.

Sometimes bands of connective tissue may be seen to appear together with the advancing atrophy of the retina, as in Fig. **36**.

A description of these that is generally applicable cannot be given.

*Question 5*

## In how far can a Conclusion be Drawn from the Ophthalmoscopic Picture Concerning the Etiology of White Spots?

*Differential Diagnosis of White Spots from the Etiological Standpoint*

As the etiology of the white spots may vary greatly, and as, on the other hand, the cause of the spots cannot be ascertained in this way alone, the final decision as to the etiology must be left to the results of a general examination. It must always be borne clearly in mind that the eye is not an organ standing alone by itself, but that it is a part of the entire body, and that, with few exceptions, the diseases of the fundus are simply symptoms of general diseases. It is as true of white spots as it is of hemorrhages that they are signals of warning to show us that danger threatens. The retina, with its very complicated structure and its highly developed functions, is a specially fine reagent to a great many disturbances of the general organism.

*Our duty is therefore imperative, just as it is in cases of hemorrhage into the retina, to make a thorough examination of the body, particularly of the urine and of the blood. If nothing is found on the first examination, something may be detected on the second, for the change in the retina precedes the other symptoms in many cases, but it is rarely misleading.*

*Von Michel* frequently told of a case in which he made the diagnosis of albuminuric retinitis, although the family physician could discover no albumin in the urine in spite of repeated examinations; it was not until after the urine had been precipitated that some granular casts were found, and yet

the patient died two years later with the symptoms referable to a contracted kidney.

What was said above, that in the determination of the etiology of a disease of the retina reliance must be placed exclusively on the results of the general examination, must not be taken literally, for the expert can certainly make the etiological diagnosis in many cases from the ophthalmoscopic picture—provided that the change in the retina is typical. This is, unfortunately, not always the case, so the statement generally holds good, even for the expert.

We will now try to ascertain what characteristics are peculiar to the individual forms, but it must be stated again that, of the whole number of changes described, none may perhaps be present, except one or two minute white spots, or a small hemorrhage. We will first divide the cases, according to an outward clinical symptom, into

## (a) Bedridden, Febrile Patients (Retinitis Septica)

The internist knows that typhoid fever, miliary tuberculosis, tuberculous meningitis, and sepsis can be differentiated with the ophthalmoscope. Let us suppose that we have been called into consultation over such a patient, who has high fever and no characteristic symptoms. What is to be expected from us?

In sepsis we find in the fundus near the papilla, never in or near the macula, medium-sized, roundish, or oval white spots, and similar hemorrhages, but they do not lie regularly in the vicinity of the vessels, as in thrombosis. In many cases the hemorrhages are very large and extensive, often so as to cover the blood vessels.

In typhoid fever we never find such white spots and rarely hemorrhages. It is self-evident that these may sometimes be absent in sepsis, so it is only the positive condition that is diagnostic.

In tuberculous meningitis we generally find an optic neuritis, like that shown in Fig. **81**.

In miliary tuberculosis, on the contrary, we find yellowish, or yellowish gray spots, as represented in the same picture, and as they have been described under tubercle of the chorioid (page 180). The picture was taken from a case of miliary tuberculosis which proved on autopsy to be also one of a severe meningitis.

The importance of ophthalmoscopy in the differential diagnosis of the above diseases has been decreased a good deal by the introduction of such specific reactions as that to tuberculin and *Widal's*.

## (b) **Nonfebrile Patients**

**1.** *Retinitis albuminurica.* This comes first in importance. The beautiful picture of the *stellate figure in the macula* (Fig. **34**) *is so impressed on the minds of most students that they expect to find it in every case of this disease, and yet it is met with only exceptionally; ordinarily we see only single white spots of the form and size depicted in Fig.* **31.**

The *characteristic* signs of an albuminuric retinitis are as follows:

($\alpha$) Signs on or about the papilla, such as an optic neuritis with a large or a small white ring (Figs. **32** and **33**), but these may be absolutely absent, as in Figs. **30** and **31.**

($\beta$) Hemorrhages, both striated (Fig. **32**) and punctate (Fig. **31**). These likewise may be lacking in any given case (Fig. **30**).

($\gamma$) Little white spots in and about the macula (Figs. **30** and **31**). These also may not be present.

($\delta$) The stellate figure in the macula (Figs. **30** and **34**), but this is by no means always present and it may be met with in other diseases.

These pictures are shown for the purpose of demonstrating that those things which give the characteristic appearance to one picture may be completely absent in another, and that the same cause may give rise to the most diverse pictures. An idea of the protean character of this disease can be obtained by combining the different forms, strengthening (Fig. **34**) or weakening the individual factors.

There are other signs in addition to those that have been mentioned, but they are of less diagnostic importance.

($\epsilon$) Changes in the blood vessels, in the form of accompanying stripes (Fig. **33**), interruptions of the column of blood, and spindle-shaped pouchings (Fig. **34**).

($\zeta$) Detachments of the retina (Fig. **33**).

($\eta$) Changes in the vessels of the chorioid (Fig. **69**).

($\theta$) Increase of the signs on the papilla to such a degree that it is difficult to differentiate it from a choked disk with patches of degeneration (Fig. **20**). The diagnosis is finally made from the general condition (see page 82).

It still remains for us to consider the details of these signs, referring to the various pictures and the accompanying text. There is one point that needs to be brought out again, the behavior of the vessels in albuminuric retinitis; the arteries are much underfilled, while the veins, on the contrary, are distended. A choked disk may be simulated very readily by an increase of the symptoms in the optic nerve, especially when there is at the same time an œdema of its head, the more so as choked disk sometimes occurs together with these patches of degeneration in the retina. *v. Michel* used to say humorously that he knew a certain high official to be a smart fellow,

because he had made a timely recognition of the albuminuric character of an optic neuritis that had been mistaken elsewhere for a choked disk.

All of the forms which were mentioned in the description of the pathology of the white spots are to be found in the picture of albuminuric retinitis. We see the degeneration of the nerve fibers in Fig. **32,** the fatty degeneration of the supporting fibers in the stellate figure in Fig **30,** the deeply situated particles of fat in Fig. **31,** and so on. To what the enormous changes seen in Fig. **34** are due cannot be told with certainty, several processes take part there at the same time, degeneration, fatty degeneration, calcification and exudates. All of these things are to be referred primarily to vascular changes, which consist essentially of hyaline degeneration of the small and smallest arteries and of the capillaries.

Albuminuric retinitis occurs in all forms of chronic nephritis, but is particularly common in the primary interstitial form, Bright's disease, and in the arteriosclerotic form; it is met with in about 20 per cent. of all these cases. Both eyes are generally affected.

The prognosis is very grave. The probable length of life after the onset of an albuminuric retinitis is at most two or three years in 90% of the cases.

The prognosis is essentially better in cases due to the nephritis of scarlet fever and of pregnancy, in which, when the acute symptoms are not caused by an exacerbation of a chronic nephritis, complete recovery may take place, and even a detached retina may become reattached.

The question whether an abortion is justified in an albuminuric retinitis due to pregnancy, is answered in the affirmative by most writers, especially when a further loss of the vision that was impaired in the first pregnancy is to be expected in the second.

The albuminuria of eclampsia can give rise to an albuminuric retinitis only when it persists after delivery.

The amaurosis observed in uræmia and eclampsia is of cerebral origin and gives no signs in the fundus.

*Retinitis circinata* needs to be differentiated from albuminuric retinitis. Spots are seen in this disease, some isolated, some confluent, which appear quite like those in albuminuric retinitis and differ in only the one feature, that they surround the cloudy region of the macula in a large, transverse oval, about 3 or 4 P. D. across. No albumin or casts are to be found in the urine. The condition is one of sclerosis of the delicate vessels of the macula.

2. The characteristic picture of albuminuric retinitis is also met with in *diabetes mellitus*, when it depends on the same pathological condition.

Fig. **35** furnishes a good example. A stellate figure is never found in the macula and the inflammatory symptoms on the papilla are usually wanting. Diabetic retinitis is very apt to occur along with other diabetic diseases of the eye, especially with iritis. This fact is of a certain value in making the differential diagnosis from albuminuric retinitis, and so is the one that hemorrhages are generally more abundant. A number of minute

bright points can be seen in the macula by the direct method. When a central scotoma is observed in diabetes, indicating the presence of an axial neuritis, it is very small, as a rule about 5 degrees, as compared with the scotoma in albuminuric retinitis, which generally has an extent of over 10 degrees.

Diabetic retinitis is usually met with only in the worst cases of diabetes, hence its prognosis is pretty bad; half of the patients, on the average, die in the course of the next two or three years, but some live ten or fifteen years with this affection.

In its later stages diabetic retinitis frequently tends to proliferations of connective tissue, retinitis proliferans (Fig. **36**).

3. *Retinitis leucocythæmica.*— In addition to the changes described on pages 102 and 119, broadening and tortuosity of the vessels, yellow red fundus, and hemorrhages, we see in leucocythæmia whitish gray patches with bloody margins, which vary in size from quite small to as large as the papilla, situated at the equator, as a rule, more rarely at the posterior pole, and are sometimes slightly elevated. At times an optic neuritis and such brilliant fatty spots are to be met with as are to be seen in an albuminuric retinitis. Leucocythæmic retinitis is almost always bilateral and accompanies only the splenic and the myelogenic forms of the disease, never the lymphatic.

4. White spots are seldom found in *retinitis anæmica;* they are more common in cancerous cachexia, when they appear in the form of isolated, silver white, round, oval, or striated patches. Usually hemorrhages also are present. The blood vessels themselves are very bright and consequently have about the same tone of color as the rest of the fundus, as the result of which they do not stand out clearly, but seem to disappear suddenly as they leave the papilla, which is pale.

5. *Retinitis syphilitica* appears in 4 forms: 1, as a chorióretinitis (see page 179); 2, in a diffuse form (see page 149); 3, as a neuroretinitis (see page 75); 4, in the form that is pictured in Fig. **38.**

This picture resembles that of a partial occlusion of the central artery. In this particular case the condition seemed to be an occlusion of a cilioretinal artery, as a connection with the true retinal vessels could not be discovered. A striking symptom is the apparently fibrinous exudates in the vicinity of the veins and arteries elsewhere in the fundus.

This case was one of syphilis in the beginning of the second stage. The sight of such white spots must always arouse the suspicion of syphilis, and the diagnosis is then made positive by *Wassermann's* reaction.

The prognosis is not so bad when proper treatment is instituted, yet in this particular case the vision remained very badly impaired on account of the grave injury that had been done to the retina by the occlusion of the artery, which manifested itself in the form of a large central scotoma.

Proliferations of connective tissue are also seen to take place as a consequence of syphilis, as in Fig. **37** (retinitis proliferans).

6. *Retinitis proliferans.*— The essential features of this condition have already been described on page 129, under the caption of connective tissue.

7. In cases of *choked disk*, especially after the condition has lasted a long time, yellowish white spots may sometimes be found without any true retinitis. These are usually caused by patches of degeneration in the layer of nerve fibers (see page 80). The knowledge of this fact is very valuable in the differential diagnosis of this condition from the so-called albuminuric choked disk (see page 79).

# PLATE XVII

## Fig. 30.  Retinitis Albuminurica

(See page 133)

The fundus is very darkly pigmented, a purely incidental condition, and distinct reflex bands can be seen along the vessels. The papilla is normal. White striae, that form an incomplete star, are to be seen in the region of the macula. The center of the star is marked by a heap of pigment. A number of large and an enormous number of small spots are visible in the vicinity of the star.

No hemorrhages and no distinct changes in the vessels can be seen. The white spots are usually caused by fatty degeneration (see page 130).

The stellate figure is a very prominent and suggestive symptom of albuminuric retinitis, but it is by no means always present. Indeed, it is not pathognomonic of this disease, for it is met with in others, such as tumor of the brain, and syphilis, although much less commonly.

Albuminuric retinitis occurs chiefly in connection with chronic interstitial nephritis, and the patients usually die within 2 or 3 years.

## Fig. 31.  Retinitis Albuminurica

(See page 133)

This is by far the more common form of albuminuric retinitis. The changes may indeed be much more trivial and lacking in character than the ones depicted; one or two white spots, with or without hemorrhages, may sometimes be all the basis on which to make the diagnosis.

A large number of roundish, yellowish white spots are to be seen below the upper temporal artery, following fairly well along its course. A little below these are a number of spots of blood, which lie in the deeper layers of the retina, as shown by their roundish form. The entire region of the macula is covered by a slight veil, which completely hides the markings of the chorioid that are to be seen distinctly in its temporal portion. The large dark spot to be seen at the lower margin of the picture is to be looked upon as congenital (*nævus*), because of the absence of signs of inflammation and degeneration in its vicinity.

Tab. 17.
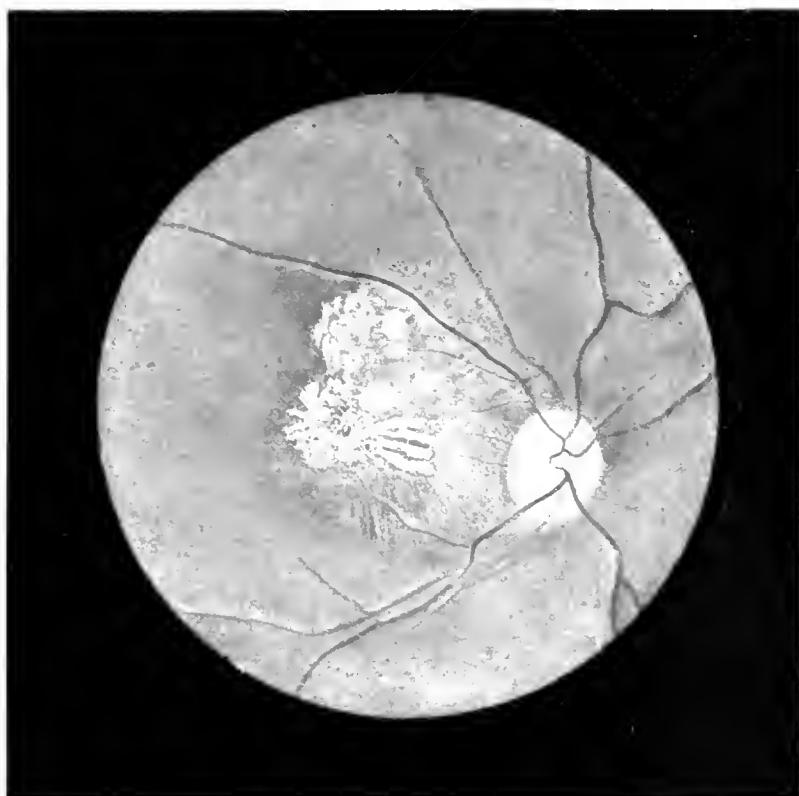


Fig. 30.
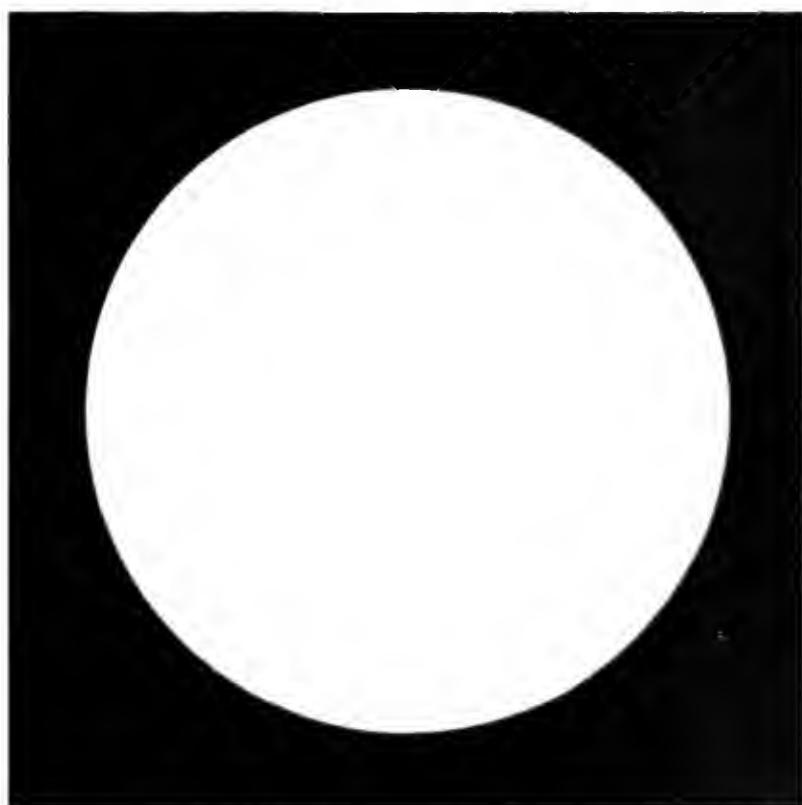


Fig. 31.

PLATE XVIII

Fig. 32. Neuroretinitis Albuminurica

Fig. 33. Neuroretinitis Albuminurica Gravidarum with
Detachment of the Retina

# Fig. 32.   Neuroretinitis Albuminurica

The morbid changes in this picture are confined wholly to the papilla and its immediate vicinity.  The papilla is very red, and is surrounded by a white ring, about a papillary diameter broad, which shows in certain places a distinct, radiating striation.  This may simulate, to the passing glance, a resemblance to medullated nerve fibers, but the changes in the vessels and the hemorrhages immediately correct such an error.  The striations show that the lesion lies in the layer of nerve fibers which radiates from the papilla. The disproportion in the degree of fullness of the arteries and of the veins indicates a serious inflammation of the head of the optic nerve that has caused a compression of the vessels.  If the head of the optic nerve were greatly swollen the picture would closely resemble one of choked disk (see page 133).

# Fig. 33.   Neuroretinitis Albuminurica Gravidarum with Detachment of the Retina

This picture has a certain resemblance to the preceding, yet no hemorrhages are present, and the form of the change is not the same.  There is a disproportion in the degree of fullness of the arteries and of the veins, just as in the preceding case.  The white stripes that accompany the vessels may be ascribed to extravasation, or distention of the perivascular lymph spaces.

The picture was complicated in this case by the presence of a large detachment of the retina.

The prognosis, as regards both the reattachment of the retina and the life of the patient, is materially better when the condition is due to the nephritis of pregnancy than when it is occasioned by other causes (see page 134).

Tab. 18.
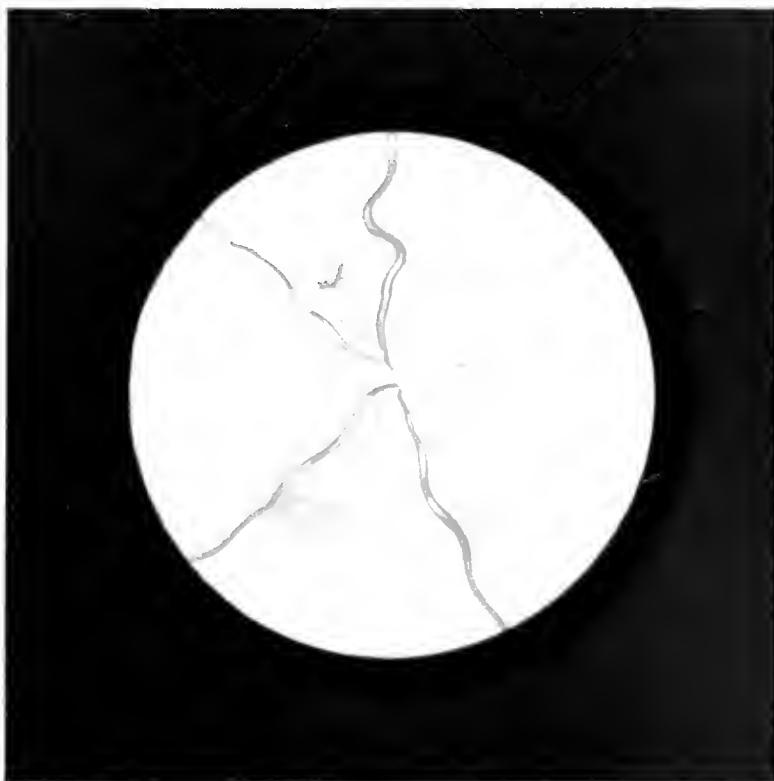


Fig. 32.
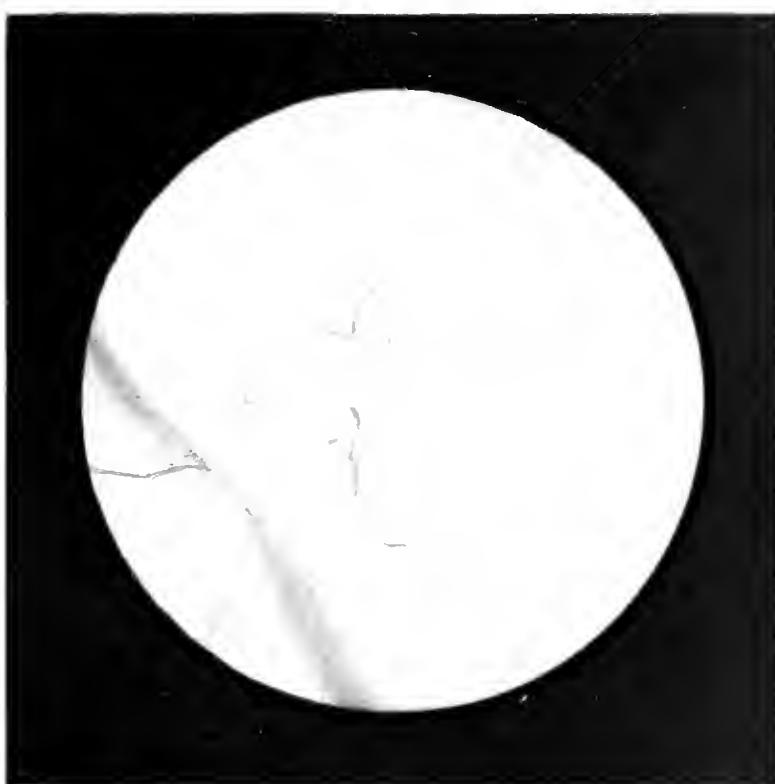


Fig. 33.

## PLATE XIX

Fig. 34.—Very Severe Neuroretinitis Albuminurica

Fig. 35.—Neuroretinitis Diabetica

## Fig. 34. Very Severe Neuroretinitis Albuminurica

(See page 133)

The entire vicinity of the optic nerve, for a distance of from two to five papillary diameters, is transformed into a chalky white layer which has, here and there, a reddish tone. A beautiful stellate figure is to be seen at the macula. The arteries are small, the veins dilated (see page 133).

This patient died four weeks after the picture was taken (see page 154).

## Fig. 35. Neuroretinitis Diabetica

(See page 134)

Diabetes can produce a picture that is quite similar to that caused by nephritis because the anatomical basis of the changes is the same. The papilla is very red, its margins slightly hazy, and there is a little œdema in its vicinity. Numerous little spots of hemorrhage can be seen in the upper part of the picture, one of which lies so close to a vessel of the retina that the latter must be supposed to be thrombosed.

The lower part of the picture exhibits many yellowish white spots, some discrete, others blended into large patches. The color of these varies with their age, the younger ones are the whiter. They become reddened and lose their sharp contour in the stage of absorption.

Tab. **19.**
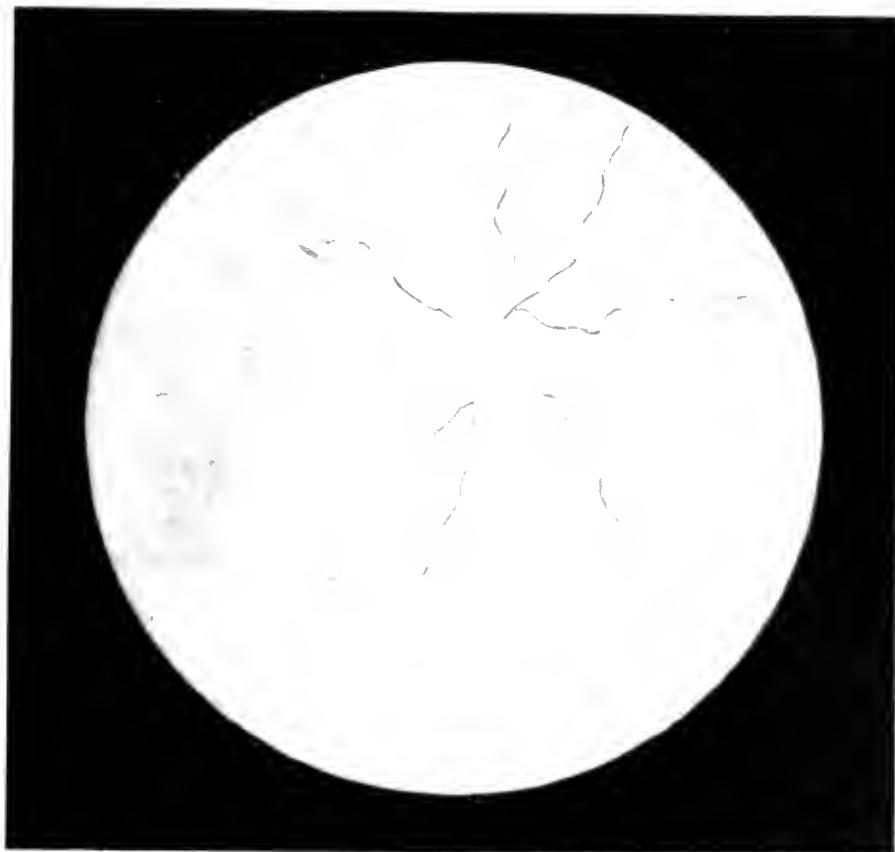


Fig. 34.



Fig. 35.

## PLATE XX

## Fig. 36.   Retinitis Proliferans in Diabetes

(See pages 129 and 134)

Retinitis proliferans not rarely is the result of a diabetic retinitis. The masses of connective tissue follow the vessels, from the adventitial sheaths of which they are accustomed to originate. Some of the vessels of the retina have disappeared, some show varicosities (see page 102).

Hemorrhages and white spots complete the picture.


## Fig. 37.   Retinitis Proliferans in Syphilis

(See pages 129 and 135)

The papilla has a peculiar, cold, red tone, which is characteristic of an optic neuritis that is undergoing involution.

Along the upper temporal artery and vein are cordlike bundles of connective tissue, which originate from the sheaths of the vessels, just as in the preceding case.

This patient presented the symptoms of syphilis in the secondary stage; *Wassermann's* reaction was positive.

Otherwise the fundus is normal, but atrophic spots may sometimes be seen in the periphery.

Tab. **20.**



Fig. 36.



Fig. 37.

## Fig. 38.   Retinitis Luetica
(See page 135)

Attention is called chiefly to an opacity which passes transversely across the field of vision, is about one papillary diameter broad, and is to be ascribed to an occlusion of a cilioretinal artery which supplies the region of the macula. The vision was greatly impaired by the presence of a central scotoma. The papilla is normal. Large exudates, that look like bits of cotton, can be seen on some of the vessels, especially the veins (see page 135).

These symptoms appeared about six months after syphilis had been contracted.

## Fig. 39.   The Same Case Six Weeks Later

The central opacity has subsided considerably. The vision is still badly impaired by the persistent central scotoma. A coronula, such as is often to be seen after occlusion of the central artery, is visible in the macula. The exudate has undergone involution to a considerable extent.

Two months later nothing was visible except the coronula and the obliteration of the vessel lying farthest to the left.

Tab. **21.**
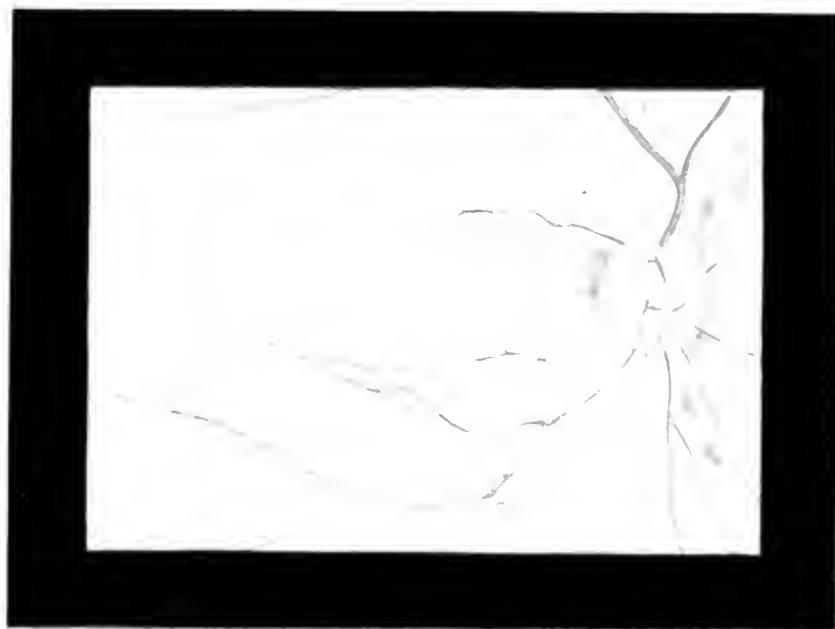


Fig. 38.
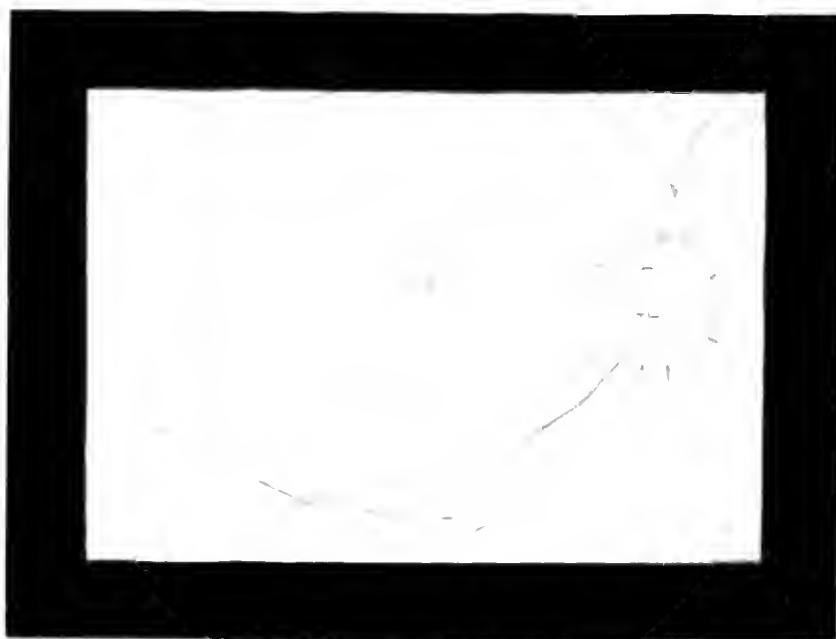


Fig. 39.

## Fig. 40.   Sympathetic Optic Neuritis and Chorioiditis

This picture was taken from the right eye of a man, 20 years old, whose left eye had been lost as the result of an injury with subsequent iridocyclitis.

About two months after the injury some deposits appeared on *Descemet's* membrane in the other eye, together with a slight iritis. The vitreous was filled with minute, diffuse opacities, yet the fundus could be seen very well. Vision was reduced to $\frac{1}{3}$ the normal, the visual field was normal, and no central scotoma could be demonstrated. The papilla was very red, its upper margins indistinct, the veins were distended, the arteries scarcely changed, and no distinct œdema was visible in the retina. Far in the periphery, moved inward a little in the picture, were several sharply defined, yellowish spots. The color about them was slightly brownish, but there was no true pigmented edge, and no lump of pigment. Such spots are thought to be retinal by many authors, yet the entire character of the disease indicates that they are situated in the chorioid. The patient was under observation for about three years, and the spots did not change during this time.


## Fig. 41.   Colloid Deposits on the Vitreous Lamella of the Chorioid

This picture was taken from the eye of an old woman. The fundus is normal, of the tesselated type. The optic nerve and the vessels are likewise normal. In the region of the macula, and in the vicinity of the papilla can be seen a number of roundish spots with a rather strong pigmentation about them. The spots near the papilla are brilliantly white, while those in the macula have a faded, reddish gray appearance. This reddish gray is not the usual color, which is commonly the same as that of the brighter parts lying near the papilla. These so-called colloid deposits are thickenings of the lamina vitrea which destroy the pigment epithelium at the places where they occur. As a rule the vision is very little disturbed (see page 127).

Tab. **22.**
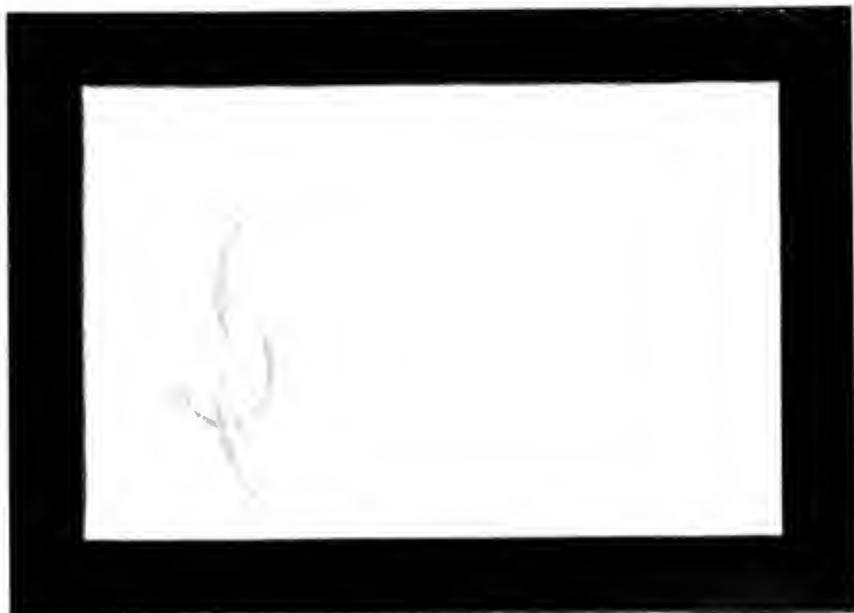


Fig. 40.



Fig. 41.

# III. DIFFUSE OPACITY OF THE RETINA

## (a) *without great differences of level,*

may be due to various anatomical conditions, the same as the white spots.

1. The commonest cause is œdema. The portion of the retina affected appears gray, or reddish gray, and the markings of the chorioid beneath it can be seen very indistinctly. The latter characteristic is particularly evident when the œdema is found in a more or less albinotic fundus (see Fig. **22**).

When, on the other hand, the color of the fundus is uniform, this is a very deceptive sign (see Figs. **18** and **31**, center). The vessels of the retina are simply covered by a thin veil, or they are embedded, so that they appear to be broken in places, according to the degree of the œdema.

*Œdema* is met with in almost all severe diseases of the retina, for example, in nearly all the cases that have been described in which white spots were to be observed, yet it is commonly cast into the background by the other, more prominent changes in the eye (Fig. **31**).

*Œdema* is also met with in circumscribed inflammations of the chorioid (see page 125). When such an œdema occurs in the region of the macula alone it is usually very difficult to recognize, and yet we are guided pretty often to this diagnosis by a fairly rapid, great impairment of the vision. The pupil must then be dilated to admit of a more accurate examination, and when the diagnosis is still not positive the test for central blue blindness should be made. This is done best by *Haitz'* method.

Œdema plays a very important part in the so-called *retinitis diffusa*. It is, in most of these cases, a local phenomenon of diffuse neuroretinitis, and appears as a sequel to any optic neuritis. It is particularly well marked in syphilitic disease of the head of the optic nerve. To a less marked degree such an œdema can be seen in Fig. **18**, in which it surrounds the reddened, indistinct papilla like a ring. The diagnostic importance of this œdema in determining whether a condition is optic neuritis or pseudoneuritis has been pointed out on page 71.

Besides the gray color we see that the vessels of the retina appear to be slightly, but distinctly veiled. The obliteration of the markings of the chorioid is not evident because the fundus is strongly pigmented. In other cases the œdema may be considerably more marked, and may have even a striated appearance when it lies chiefly in the layer of nerve fibers. This form is usually of a syphilitic nature. Hemorrhages and white spots are considerably less common then than when it is due to other diseases.

Although syphilis is the most common and the most important cause of

peripapillary œdema, it is not the only one. The affections of the optic nerve, due to diseases of the ear, are very important (see page 77). In these cases the œdema may precede the changes in the optic nerve, and may be at times the only symptom present. It seems to lie in the deeper layers of the retina, as striations are almost never seen.

It may also be observed in choked disk, especially in the albuminuric variety, but hemorrhages and white spots are seldom wanting in such cases.

2. Another, very much rarer, form of diffuse opacity of the retina is the *diffuse infiltration with white blood corpuscles*, an outspoken inflammation. This is characterized by the very marked sheathing of the vessels of the retina shown in Fig. **42**, which is to be ascribed to a distention of the adventitial sheaths of the vessels with white blood corpuscles. This form is likewise met with in syphilis, and sometimes in leucocythæmia. Considerable accumulations of leucocytes in circumscribed places make themselves evident as white spots.

3. *A necrosis of the inner layers of the retina* in connection with recurrent œdema (Figs. **44** and **45**) underlies the opacity of the retina in occlusion of the *central artery.* In these cases the demonstration of the cherry red spot (see also page 72), which is sometimes surrounded by a particularly cloudy halo, but usually lies in its opaque surroundings without such a special border, is of diagnostic value. The opacity itself is brighter in these cases than it is in purely inflammatory œdema, and sometimes it is perfectly white. The arteries are usually, though by no means always, invisible, or considerably diminished in size. The eroded place in the vessel itself can be seen in many cases as a white spot, as in Fig. **45** on the papilla. Pulsation is absent when pressure is made on the eyeball (see page 107). The opacity gradually retrogresses (compare Fig. **45** with Fig. **44**) until finally the normal color of the fundus returns, with a uniform whiteness of the papilla and an extreme contraction of the arteries, but intermixed with a gentle dark gray tone, due to a migration of pigment cells into the atrophic retina (Fig. **16**). This migration of pigment is particularly distinct in the region of the macula, where it takes part in the formation of the coronula (see page 127).

The occlusion sets in with sudden amaurosis, which commonly is irreparable.

Sometimes it is not the entire trunk of the artery that becomes occluded, but one or more of its branches (see Fig. **38**, paying no attention to the white spots). Then the amaurosis is not total, but affects only certain portions of the field of vision.

It also may happen that the trunk of the artery is occluded behind the point where certain branches are given off. The region supplied by these branches is then seen to be of a normal red color, while the rest of the retina is opaque. The vision appertaining to this place is preserved.

4. The opacity in cases of commotio retinæ (Fig. **43**) is to be looked

upon as due to *vasomotor disturbance with transudation.* The opacity may have a close resemblance to the one just described. It is usually found near the macula or the papilla. The condition of the vessels, which are normal or dilated, and the other signs of traumatism frequently to be observed, such as hemorrhages and ruptures of the chorioid, taken together with the history, generally enable a differential diagnosis to be made from other forms of opacity. Usually the vision is harmed little if at all. The transudate passes away in a few days.

5. Another cause of a diffuse opacity is the *flat detachment of the retina* (Fig. **46**). The word "flat" must be emphasized in this connection, because the chief and most striking symptom in the eye when the detachment is gibbous is the difference in level.

The color of the detached portion is gray, sometimes gray green or gray blue. The detachment may be total (Fig. **46**), or partial (Fig. **47**). Particular attention is to be paid in the **differential diagnosis** to the white cords, in the vicinity of which the vessels of the retina often show abnormal bends. These are the apices of the folds formed in the retina.

A very valuable symptom is *the absence of the markings of the chorioid* in the detached portion, which are visible elsewhere. The vessels of the retina throughout the same area are very dark and have no reflex.

The local elevation of the retina may frequently be perceived by parallactic displacement and by determination of the refraction.

In cases of total detachment of the retina the loss of vision is very great; when the detachment is partial the loss is greater or less, according to the position it occupies. The field of vision usually exhibits a contraction that corresponds to the detached part.

Ordinarily the detachment begins in the upper part of the retina and causes a defect in the lower part of the field of vision, so that when the patient looks at a person the latter may seem to have no legs; later it moves downward and causes a defect in the upper part of the field, so that the person looked at may seem to have no head.

When a detachment commences the patients complain of *subjective* sensations of light, the so-called photopsias, which are described as scintillations, flashes, balls of fire, sparks, or circles. Objects also appear to be distorted, bent, or jagged (metamorphopsia), and sometimes they seem to be of a pronounced green. The detached places are blind to blue, i.e., blue is perceived by them as green or gray, and the patients suffer from hemeralopia, i.e., their vision becomes disproportionately bad as the light is reduced.

The **etiology** varies. It may be

1. purely ocular, or local, as when the detachment is caused by an injury, either a perforating wound, or a contusion, hemorrhages, tumors, or a high degree of myopia;

2. general, as when due to nephritis, arteriosclerosis, or syphilis. In many cases the diagnosis of these diseases as the cause can be determined from

other ocular signs, as from sclerosed vessels, white spots, or black spots, but in others the differentiation must be made by means of a general examination. Arteriosclerosis is the etiological factor in Fig. **47,** as shown by the sclerotic vessels in the vicinity of the papilla.

(b) *Diffuse Opacity of the Retina with Marked Differences of Level.*

A difference of level is indicated when some parts of the fundus are plainly visible during an ophthalmoscopic examination, while others are indistinct. Detachment of the retina shares this symptom with quite a number of other diseases, such as a great amount of œdema, and opacities in the vitreous, but it is characteristic of detachment of the retina when it disappears as soon as the observer makes his examination from a greater distance than usual, and the portions that were indistinct become clear, while those which were clear at first become indistinct.

The reason of this phenomenon is that the detached portions of the retina lie farther forward than the rest and consequently have a different refraction. In most cases the diagnosis of a detachment of the retina can be made more conveniently by simply casting light into the eye with the mirror of the ophthalmoscope than by an examination of either the inverted, or the upright image, as the detached portions then look much less red than the others.

When the detached retina lies very far forward it may sometimes be seen by oblique illumination.

Figures **48** and **49** therefore do not give pictures that are true to nature of a detachment of the retina, they are, rather, composite pictures which assume the changes to be made in the position of the examiner.

Figures **48. 49.** and **50** exhibit types of such a detachment.

1. Fig. **48** shows a *gibbous detachment caused by an exudate.* The arching of the retina can be seen, in addition to its changed color and the formation of folds. The papilla has a slight haziness, which might perhaps cause it to be mistaken for an optic neuritis (see page 72).

A distinct movement of the bulla back and forth may be seen during movements of the eye.

2. Fig. **49** shows, in contrast to the above, *a detachment of the retina caused by a tumor of the chorioid.* Its margins are sharply defined. No movement can be detected; on the contrary, the detachment gives the impression of a solid, firm mass, which is increased by the fact that a reddish shimmering from the tumor beneath the detached retina can be seen in certain places.

The difference is not so distinct as it is in these pictures in many cases, and then the differential diagnosis is best undertaken with the aid of *Hertzell's* lamp, which, when introduced into the mouth, transilluminates the globe from behind. A dark spot is to be seen when a tumor is present, while a serous effusion allows the light to pass through freely. This method is particularly useful when tumors are situated in the posterior segment of the eye-

ball, while for those in the anterior segment better service is obtained from *Sach's* lamp, which transilluminates the eyeball laterally.

3. Fig. **50** shows a *glioma of the retina.* The diagnosis rests chiefly on the facts that an embedding of the vessels of the retina can be seen in the tumor, and that the patient is young.

In a total, funnel-shaped detachment of the retina no picture of the fundus can be obtained, as can readily be understood if we look at Fig. R. If the lens is transparent the detached retina can often be seen by oblique illumination, and the rounded protrusion can sometimes be seen by simply throwing light into the eye with the mirror of the ophthalmoscope, but when these means fail a conclusion as to the condition of the retina can be drawn from the projection, as a large hemorrhage into the vitreous may produce a similar picture. Loss, or limitation of the projection is indicative of detachment, correct projection of a hemorrhage.

Fig. R.

## Concerning the Prognosis as to Life of Diseases of the Retina and Chorioid

*Geiss* has drawn conclusions, of which the following is an abstract.

### I. *Arteries of the Retina.*

1. *Marked Arteriosclerosis of the Retinal Vessels.*

All of the patients observed, to the number of 17, ranging in age from 40 to 70 years, suffered from an attack of apoplexy within 4 years at the most.

2. *Sudden Occlusion of the Central Artery.*

(a) Seventeen patients, between 40 and 70 years of age, with arteriosclerosis, but without heart disease. Of these 14 had an attack of apoplexy within 2 years, the other 3 died from arteriosclerosis in from $1\frac{1}{2}$ to 7 years.

(b) Six patients, between 40 and 70 years of age, with *heart disease.* One died of apoplexy 3 years later, 4 from heart disease within 2 or 3 years, and one was still alive at the end of 4 years.

(c) Nine patients, 39 years or less of age, with *heart disease.* Prognosis not so bad.

3. Syphilitic diseases of the retina do not have the same bad prognosis as arteriosclerotic changes.

### II. *Veins of the Retina.*

Thrombosis of the veins of the retina has not the bad prognosis of the diseases of the arteries. It is a purely local disease in 50% of the cases, and in only 50% is a forerunner of a sclerosis of the cerebral vessels, which may not make itself manifest until a long time afterward.

### III. *Vessels of the Chorioid.*

No conclusions as to the condition of the vessels in the brain can be drawn from sclerosis of those in the chorioid.

### IV. *Retinal Hemorrhages*

in arteriosclerosis, diabetes, and chronic nephritis are, as a rule, harbingers of hemorrhages into the brain, which yet may not occur until after the lapse of years.

Hemorrhages into the vitreous in young persons, isolated hemorrhages in the macula, and the retinal hemorrhages caused by syphilis do not partake of this bad prognosis.

### V. *Retinitis Albuminurica.*

Of 38 patients, 29 died within 1 year; 4 died in from 1 to 2 years; 2 died in from 2 to 4 years.

Three patients with retinitis albuminurica gravidarum recovered.

### VI. *Retinitis Diabetica.*

Diabetic retinitis has a different prognosis from the isolated hemorrhages in the retina met with in diabetes, which are to be considered as precursors of cerebral apoplexy. Apoplexy supervened in only $\frac{1}{3}$ of the cases. Half of the patients died within 2 or 3 years.

PLATE XXIII

Fig. 42.   Retinitis Luetica

Fig. 43.   Commotio Retinae, or Berlin's Opacity

# Fig. 42.  Retinitis Luetica

Primary syphilitic retinitis, when not a local phenomenon of a neuro-retinitis, is a fairly rare disease in comparison with the secondary syphilitic diseases of the retina due to an affection of the capillaries of the chorioid. In the case presented here the papilla is comparatively little involved; its color is almost normal, its margins are fairly distinct, but the vessels that lie upon it exhibit a distinct obscuration and sheathing. The retina, on the contrary, is cloudy over a large extent, and only a few of the vessels of the chorioid can be seen through the opacity. The vessels of the retina look hazy throughout the entire extent of the opacity and are partly sheathed everywhere. No hemorrhages are visible; only a few, striated, whitish spots can be seen. In a similar case, which was studied pathologically by *Bach*, partial and circular inflammations of the adventitia and intima, together with obliterations of the capillaries, were found to be the cause of the trouble.

# Fig. 43.  Commotio Retinae, or Berlin's Opacity

This opacity received its name not from the city, but from the ophthalmologist *Berlin*, who was the first to describe the condition, and to differentiate it from detachment of the retina. The condition depicted here was produced by the blow of a ball against the eye of a boy 12 years old; the picture was taken a few hours after the injury.

The papilla is much reddened, its margins are slightly hazy, enough so as to be suggestive of a commencing optic neuritis, but the vessels, especially the veins, are normal. A light gray ring, with a rather darker center, can be seen in the region of the macula. Such a lesion can be mistaken for a commencing detachment of the retina when the vessels do not run smoothly over it and there is any sign of a fold. The changes, which caused only a slight impairment of the vision, disappeared completely after a few days (see page 72).

The color of the opacity may be more intense than it is in this case, it may incline more to yellow, or to white, and it need not be situated exactly in the region of the macula.

The nature of this change has not yet been learned.

Tab. **23.**
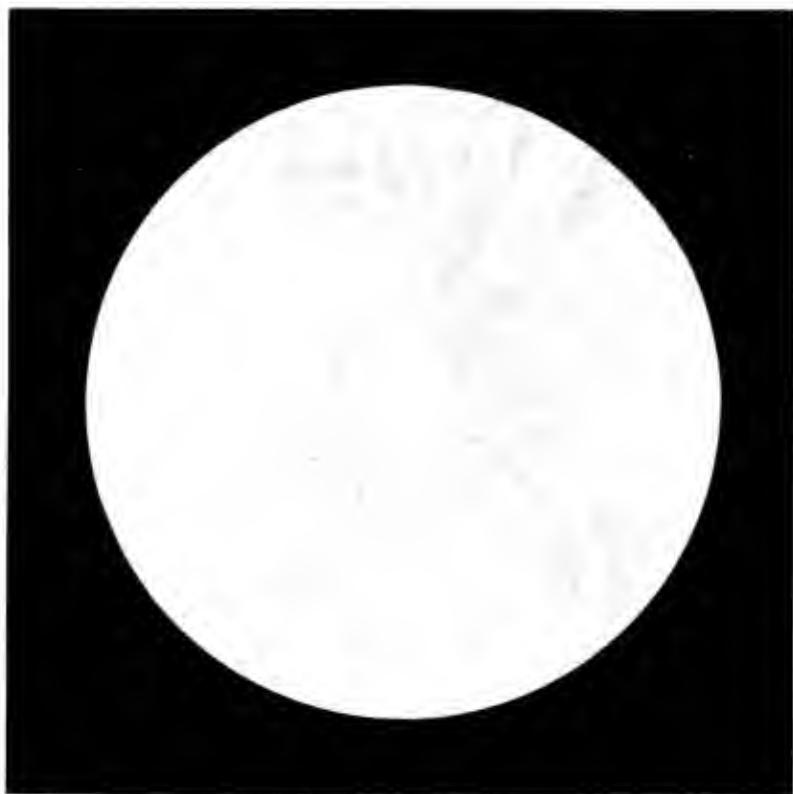


Fig. 42.
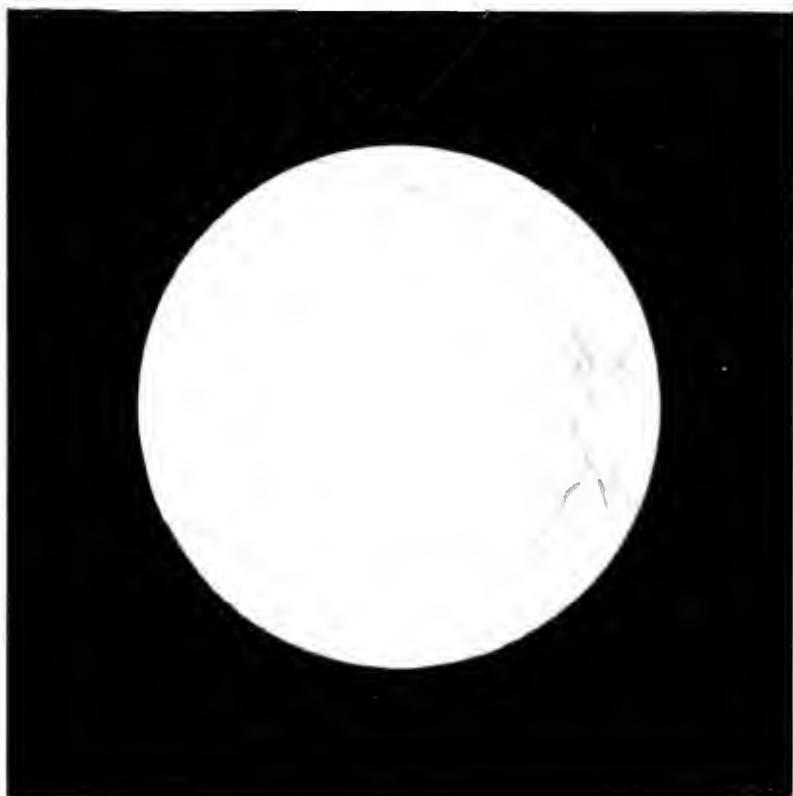


Fig. 43.

PLATE XXIV

Fig. 44.  Sudden Total Occlusion of the Central Artery, the
So-called Embolism

Fig. 45.  Occlusion of the Central Artery in a Later Stage

## Fig. 44.   Sudden Total Occlusion of the Central Artery, the So-called Embolism

Occlusion is caused by a true embolus only in extremely rare cases, and is usually due to a slowly developing, but suddenly becoming total, closure of the lumen of the artery by a proliferation of the intima, endarteritis proliferans.

The papilla is very red, its margins completely hidden, the arteries can scarcely be seen. A large area of the fundus, about 6 P. D. across and including both the papilla and the macula, is whitish gray and opaque. The well-known cherry red spot can be seen in the macula. In this case it is surrounded by a white areola (see pages 72 and 119), but this is not always present.

The white color of the fundus is the consequence of a rapid necrosis of the cerebral layer, which receives its nutrition through the central artery, in combination with an œdema. The red spot is brought out by the fact that the cerebral layer is absent at the fovea, so that the œdematous tissue is wanting and the dark color of the fovea stands out in marked contrast to its surroundings.

The arteries gradually refill through the mediation of *Zinn*'s arterial plexus (see page 168), and the minute branches in the vicinity of the macula become strikingly prominent (Fig. **45**), but the retina remains incapable of performing its functions and the papilla atrophies (see Fig. **16**).

## Fig. 45.   Occlusion of the Central Artery in a Later Stage

The margins of the papilla have become in part sharply defined again, its redness has passed away. The arteries have become refilled. The obliterated place can be plainly seen in the lower artery on the papilla. The white color of the fundus has passed off to a considerable extent. The cherry red spot is no longer so conspicuous, and there are signs of the coronula, which can be seen distinctly in Fig. **16**. An area attached like a wing to the papilla has regained approximately its normal color.

The vision remained lost in spite of the improvement in the objective symptoms.

Tab. **24.**



Fig. 44.



Fig. 45.

## Fig. 46. Flat Detachment of the Retina

The papilla is normal in every respect, as regards its color, margins, level, and vessels.

The fundus has, instead of its normal reddish color, a green gray appearance with bright and dark bands here and there. The vessels have many little tortuosities, such as are scarcely to be seen in any other condition. An artery makes a marked bend as it passes over the very white band. The entire picture seems quite dull, because the vessels have no light streaks, as is almost always the case in detachment.

This is a picture of an almost total, but flat detachment of the retina. The detachment does not extend quite to the papilla, for if it did the margins of the latter would be obscured, and then the picture would look like one of optic neuritis (see page 72).

Naturally the vision is badly impaired.

## Fig. 47. Partial Flat Detachment of the Retina

The papilla is normal. The retina in its vicinity presents discolored islands over which the vessels pass with a distinct bend. Outward and upward from the papilla is a large discolored place, which shows plainly several folds, over which the vessels of the retina deviate from their courses. On the other side of the papilla the retina is still attached and allows the markings of the chorioid to be seen distinctly through it. Some of these vessels are sclerosed, so we will make no mistake if we diagnose this as an arteriosclerotic detachment of the retina. The retinal vessels have no light streaks, except in the portions that lie on the papilla.

Tab. **25.**



Fig. 46.
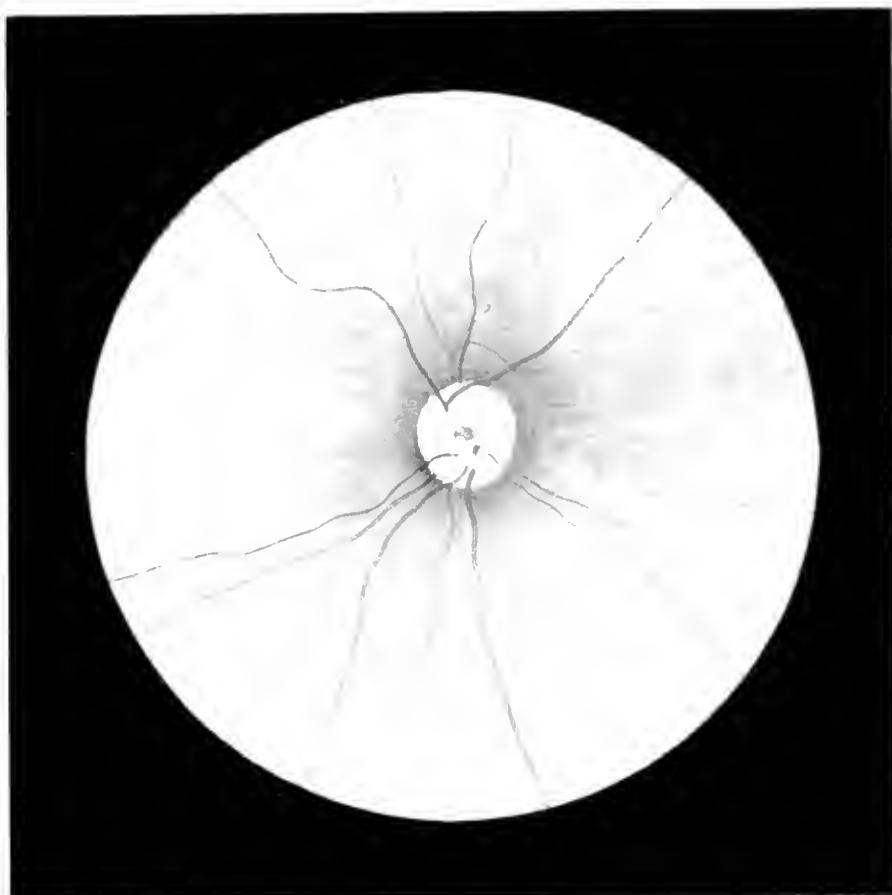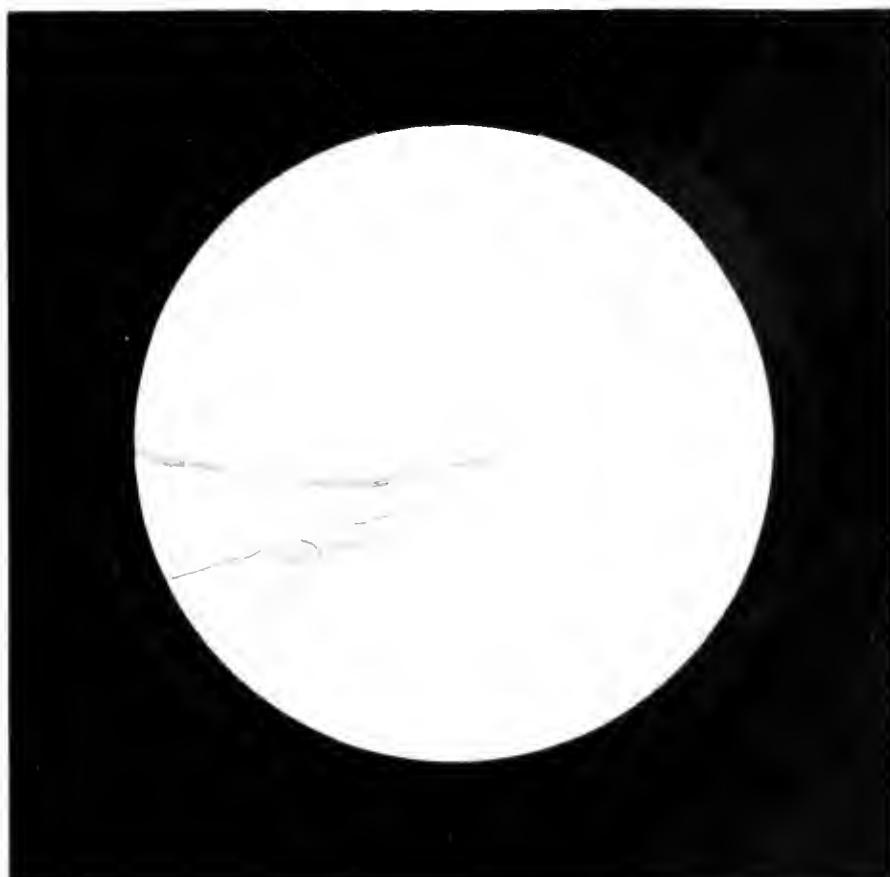


Fig. 47.

PLATE XXVI

Fig. 48.  Large Gibbous Detachment of the Retina

Fig. 49.  Detachment of the Retina Caused by a Tumor of the
Chorioid

## Fig. 48. Large Gibbous Detachment of the Retina

(See page 152)

This is a composite picture, which depicts what the observer may see at varying distances from the eye of the patient. When he focusses on the papilla the detached portion of the retina will be obscured, and, on the other hand, when he leans backward in order to see distinctly the detached portion, the region of the papilla becomes indistinct.

The margins of the papilla are not quite sharply defined. On its temporal side is a distinct conus.

The vessels of the retina are in part accompanied by broad, white bands, which have been produced by transudates that, in turn, are due to an existing nephritis.

The detached retina protrudes very far forward, shows depressions and elevations, and exhibits distinct wavy movements whenever the patient moves his head. These are to be ascribed to the fluctuations of the fluid accumulated behind the retina.

The detached portion was clear on transillumination with *Hertzell's* lamp.

## Fig. 49. Detachment of the Retina Caused by a Tumor of the Chorioid

(See page 152)

This is also a composite picture.

Although it has a certain resemblance to the preceding, yet it presents certain characteristic differences. The line of demarcation of the detached portion is quite sharp. The surface is smooth, tensely stretched, and in certain places the reddish color of the tumor can be seen shining through the retina over it. When the eye was transilluminated with *Hertzell's* lamp the region of the detachment appeared as a dark shadow.

No fluctuating movement could be seen when the head was moved.

Tab. **26.**
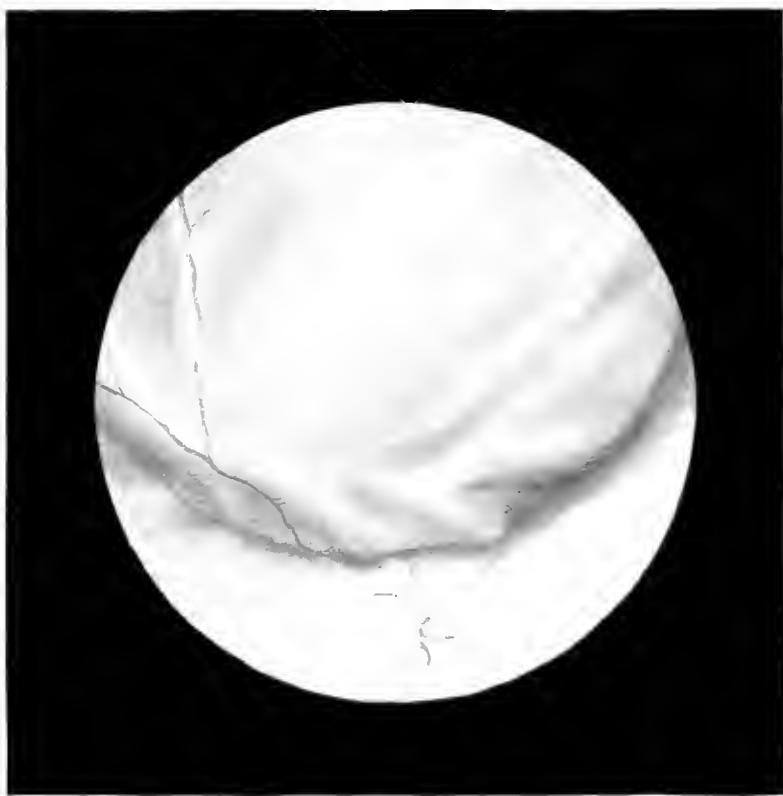


Fig. 48.
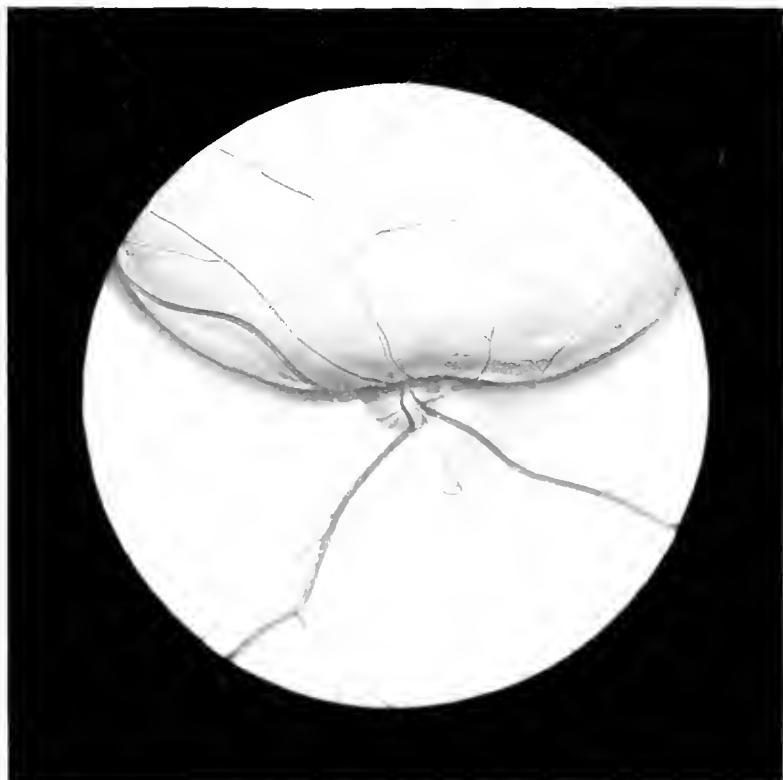


Fig. 49.

PLATE XXVII

Fig. 50.  Small Glioma of the Retina

# Fig. 50.  Small Glioma of the Retina

(See page 153)

Such a picture as this is rarely to be seen, because glioma occurs in early childhood and is not noticed in most cases, on account of the absence of subjective complaints, until nearly the entire space of the vitreous has been filled by it so as to produce the so-called amaurotic cat's eye. But, as the tumor frequently appears in both eyes, it occasionally happens to those who habitually examine both eyes with the ophthalmoscope that they are able to see a glioma of the size here depicted.

The tumor apparently starts from the vicinity of the papilla, which is overlaid by it, and measures $3\frac{1}{2} \times 4\frac{1}{2}$ P. D., i.e., $5\frac{1}{4} \times 6\frac{3}{4}$ mm. It is therefore in reality about as large as a pea. This picture was taken in January, 1910; in March the tumor was as large as a bean, by October it had filled the entire vitreous, in December, 1911, it broke through the eyeball, and then the proliferation advanced very rapidly, so that at the time of the death of the child, in May, 1912, the tumor was as large as an apple and protruded from the orbit.

The similarity of the picture to the preceding ones is very great, but it is to be noticed that some of the vessels of the retina lie in the tissue of the tumor itself. At the upper pole of the tumor may be seen several black spots and one large patch of atrophy, which indicate a simultaneous disease of the chorioid; whether or not this had any etiological connection with the tumor could not be determined. The hemorrhages near the papilla are uncommon. The bright zone about the tumor shows that the pigment epithelium of the retina is also involved.

Tab. **27.**



Fig. 50.

Chorioid

# Chorioid

## Preliminary Remarks on the Anatomy

The chorioid is rightly named, for it consists essentially of vessels which furnish nutrition to the macula and the outer layers of the retina.

On account of the great abundance of blood vessels the thickness of this membrane varies in proportion to the degree to which they are filled, and varies also in different places, from 0.05 or 0.08 mm, at the place where it passes over into the ora serrata, to 0.1 or 0.2 mm about the posterior pole.

It is very loosely connected with the sclera (Fig. **54**, S) through

1, the lamina suprachorioidea (Su). The space between the two membranes, which is demonstrable only under pathological conditions, is known as the perichorioidal space. This lamina contains many pigment cells and elastic fibers, but no vessels. Then comes

2, the lamina vasculosa, the layer of the larger vessels. Next is

3, the choriocapillaris, the layer of capillaries, which is of the greatest importance in the pathology of the diseases of the chorioid and the retina. Adjoining this is

4, the lamina vitrea, also called the lamina elastica and the lamina basalis. Finally comes

5, the layer of pigment epithelium, which appertains to the retina (see page 111), but must be mentioned in this place on account of its intimate pathological relations to the chorioid.

The *vascular supply* is through the so-called ciliary vessels, which come from the ophthalmic artery. They consist of from 4 to 6 short, and 2 long posterior ciliary arteries, which enter the eyeball near the optic nerve, and 4 anterior, which enter near the limbus. The anterior ciliary arteries run first in the four recti muscles, which they supply, and divide, each into two branches, before they reach the limbus.

The short posterior arteries branch very quickly after they have passed through the sclera and the lamina suprachorioidea, and form the main part of the arteries of the chorioid.

The long posterior arteries pass without branching in the layer of large vessels to the ciliary body, where they empty into the circulus arteriosus iridis together with the anterior ciliary arteries, but before doing so they give off recurrent branches which unite with the capillaries of the short posterior arteries.

The short posterior ciliary arteries likewise form a circulus arteriosus in

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the sclera, which surrounds the papilla and forms a connection between the ciliary and the retinal vessels. This is known as the circulus arteriosus nervi optici, or the circle of *Zinn*. This connection is of but little practical importance because it never happens that a central artery of the retina which is obstructed in its central portion is sufficiently supplied with blood through this means.

The chorioid may be divided into two very unequal portions, according to the arterial supply, the posterior and larger of which extends from the papilla into the region of the equator, the anterior from this point to its transition into the ciliary body. The former is supplied by the short posterior ciliary arteries, which therefore form the principal source of the nutrition of the chorioid, while the second receives its blood through the recurrent branches of the long posterior ciliary arteries.

The anterior ciliary arteries are of importance to the chorioid only in so far as they unite with the long posterior, through the circulus arteriosus iridis major, and thereby with the short posterior ciliary arteries, but this indirect connection with the nutrition of the globe is of practical value, for it is able to preserve the latter when all of the posterior arteries have been divided, as when the retrobulbar space has been exenterated in the removal of a tumor.

The equatorial portion has the poorest supply, which comes only through the terminal filaments of the short posterior, and of the recurrent branches of the long posterior arteries. It is therefore not an accident that this portion is the first, or the main one to be affected when degenerative processes take place in the eye, as in chorioretinitis pigmentosa, and hereditary syphilis.

The venous outflow of the chorioid is quite different from its arterial supply, as its veins carry away not only the blood from the chorioid itself, but also that from the ciliary body and the iris. Consequently they are far more numerous than the arteries and have many more anastomoses. They commonly pass from the chorioid into the sclera behind the equator of the eye in the form of from 4 to 6 large vessels. In rare cases they end at the posterior pole (Fig. 3), principally in eyes that are highly myopic.

The chorioid contains besides vessels many collagenous fibrils and elastic fibers, as well as a great quantity of chromatophores laden with pigment. The latter are to be found in all of the layers of the chorioid, with the exception of the choriocapillaris, and naturally of the lamina vitrea, but especially in the spaces between the vessels.

## General Diagnosis of Diseases of the Chorioid, so far as They are Caused by Diseases of the Vessels

The schematic drawing, Fig. 54, is, with r material changes, the same as that used by *Krueckmann* in *Axenfeld's* text-book.

The pictures in the circles show typical changes that are to be observed

in diseases of the vessels of the chorioid. A glance at the succeeding plates, which are not schematic, for example, at Fig. **74,** reveals at once a resemblance to the disturbances here delineated schematically.

Near each change is presented the corresponding microscopical picture. The retina is absent from all except Number VI, for this must be understood to be changed in all, with the exception of Number I of course, because, as has been said repeatedly, the outer layers of the retina receive their nutrition from the chorioid, and disturbances in the vessels of this membrane must naturally manifest themselves through nutritive derangements of the corresponding portions of the retina.

I. Picture I shows ophthalmoscopically a perfectly normal condition, which is likewise normal microscopically. S indicates the sclera; Su the lamina suprachorioidea; V the layer of large vessels; Ch the choriocapillaris; L the lamina vitrea; E the pigment epithelium of the retina, or at least its layer of basal cells. The latter is in this example perfectly uniform and not transparent, so that the chorioid cannot be seen.

II. In picture II the pigment epithelium is destroyed to a considerable extent as the result of disease of the subjacent choriocapillaris; it is made homogeneous, if one may use such an expression. In consequence of the breaking down of this layer the larger vessels of the chorioid can be seen in the form of a relatively bright network on a dark background, because of the pigment that lies between them. They appear to be of a normal color and they are found to be normal under the microscope.

III. The process has extended farther. The pigment epithelium and the choriocapillaris have completely disappeared. Portions of the larger vessels also are obliterated (A) (a), and consequently appear in the ophthalmoscopic picture as white cords. The vessel B on the other hand is in a normal condition.

IV. This presents a still further advanced stage. The vessels of the chorioid are almost totally obliterated (C), the vessel D alone still contains a slender column of blood within its thickened walls.

V. The chorioid has almost wholly disappeared; traces only of the intravascular pigment can be seen. As there is no pigment in the places formerly occupied by the chorioidal vessels the spaces formed by their absence have their forms and courses. When these traces of pigment disappear the pure white sclera is laid bare.

VI. This shows the way in which the black spots are brought about. Here, as in II, the choriocapillaris has been destroyed, but at the same time bands of tissue have been formed which have in their spaces newly formed pigment cells, and as these lie one behind another they give ophthalmoscopically the impression of a dark, black spot. Further, the picture shows the secondary pigmentation of the retina that appears in diseases of the chorioid.

This is brought about by the blending of the processes of the pigment cells with the glia tissue, especially with *Mueller's* supporting cells, so that

the brown granules of fuscin *penetrate into the retina.* It is self-evident that *newly formed* pigment epithelium can also proliferate into the degenerated chorioid.

*What we learn from these schematic pictures is that not only abnormal pigmentations, but* **depigmentations** *as well, are to be looked upon as signs of disease of the chorioid.*

It may be emphasized again that pigment is to be seen normally in the fundus in two places:

1. *The pigment layer of the retina,* which covers the chorioid and limits the view with the ophthalmoscope. It is only when the nutrition of this layer is impaired by a disease of the choriocapillaris, and is thus caused to atrophy, that the markings of the chorioid can be seen. But it must be remembered that similar pictures may be brought about by a congenital partial or total absence of this pigment layer, as in albinism. In such cases, however, there is none of the abnormal *heaping* of pigment and disease of the vessels that is to be seen in every pathological case. Compare the periphery of Fig. **57** with Fig. **8.**

2. *The pigment of the chorioid,* which lies in the spaces between the vessels of this membrane and may last very long in spite of serious chorioidal disease. The general statement may be made that an *abnormal heaping of pigment takes place at the same time wherever pigment is destroyed.* Pigment can migrate into the retina only when the lamina vitrea has been injured, hence it is that colloid deposits can frequently be seen on this lamina (Fig. **41**) in association with pigmentations of the retina.

*Etiology of Chorioiditis.*

*Almost all diseases of the chorioid are symptomatic of general diseases,* with the exception of those that are due to traumatism and some conditions that are congenital. Therefore a very thorough *general examination* is indicated in all such cases, just the same as in retinitis. Although certain conclusions can be drawn in a number of cases from the position, color, size, and appearance of the lesion, yet the etiology must be determined mainly by such an examination. This is particularly true for the general practitioner, who can see the black spots with the ophthalmoscope, but may not perceive or interpret the minuter differences, on account of his lack of special practice. The following proposition is particularly applicable to him: *If black or white spots are present in the fundus a thorough examination must be made of the organism, which is not to be confined to the ordinary physical and chemical methods alone, but in which the tuberculin and Wassermann tests are to be made.* A guide to the etiology is frequently to be found in other ocular symptoms: for example, if the cornea is investigated with the binocular loupe and deeply situated vessels, or fine, central, parenchymatous opacities are found, which indicate a past interstitial keratitis and may persist for years after the subsidence of such an inflammation, we have obtained an almost

positive proof of the syphilitic origin of the disease. Dots of pigment in the pupils of young persons, which are to be considered as traces of a bygone iritis, point toward the same etiology, because iritis is usually of syphilitic origin in young persons.

The following conclusion can be drawn from the ophthalmoscopic picture if we look for changes in the vessels.

*If sclerosed vessels are present in the chorioid the probable cause of the disease is either syphilis, arteriosclerosis, or nephritis; if no such vessels are present the probable cause is tuberculosis.*

In a small number of cases the disease in the chorioid is of a metastatic pyæmic nature, but these are usually associated with a simultaneous disease of the iris and ciliary body, and, as these ordinarily induce a great opacity of the refractive media, the lesions in the chorioid are not to be seen with the ophthalmoscope as a rule. Consequently they do not come into consideration in this place.

# DIAGNOSIS

## A. General Diagnosis

*After the diagnosis of a disease of the chorioid has been made the following points need to be taken into account in order to establish its etiology:*

*1, the position of the lesions in the chorioid;*
*2, the sort of pigmentation, or depigmentation;*
*3, whether changes are present in the vessels or not;*
*4, the form of the change in the chorioid;*
*5, the differences of level.*

Although we cannot deduce the etiology from the ophthalmoscopic condition alone in all cases, but very often have to rely upon the general examination, yet we are quite frequently able to do so by the observation of the following points:

*1. The Position of the Lesions in the Chorioid.*

It may perhaps seem rather forced and schematic to classify changes in the chorioid according to the position they occupy in the fundus, but it is not, for the spot appears in this or that place in accordance with certain pathological reasons. For example, the branchings of the vessels of the chorioid are most extensive in the region of the equator and the movement of the blood at this place is normally rather slow, as has been mentioned already, so if we imagine a general contraction of the capillaries to have taken place through a hyaline degeneration of their walls, we will expect the first and most prominent symptoms to appear just at this place.

Hence we differentiate between

(*a*) the periphery of the fundus, the region of the equator
(*b*) the region of the macula,
(*c*) the region of the papilla,
(*d*) disseminated distribution.

*2. The Sort of Pigmentation and Depigmentation.*

With regard to the form of the pigmentation we differentiate

(*a*) a regular, bone corpuscle form, as in Fig. **51**;
(*b*) a lumpy form, which often appears as a circle (Fig. **53**);

(*c*) a powder form, as though snuff had been sprinkled over the fundus (Fig. **63**):

(*d*) pigmentation along the vessels of the retina (Fig. **57**);

(*c*) an irregular form.

We meet with the following typical forms of depigmentation:

(*a*) a diffuse depigmentation, chiefly found in the periphery (Fig. **57**);

(*b*) a depigmentation in the form of minute spots, as in Fig. **55;**

(*c*) depigmentation in medium-sized spots, as in Fig. **79;**

(*d*) irregular.

*3. Are Changes Present in the Vessels or Not?*

Vascular changes, especially of the large vessels, ordinarily give a beautiful, characteristic picture (Fig. **64**).

In many cases changes in the vessels cannot be seen, but their presence may be inferred. For example: Diseases of the choriocapillaris lead to decolorizations of the pigment epithelium; consequently we are justified in the conclusion that there is a disease of the choriocapillaris whenever we see such depigmentations with the ophthalmoscope (see Fig. **57**). Another example: When the vessels of the chorioid sclerose and gradually become destroyed, we can often recognize the positions they formerly occupied from the fact that the pigment lying between them outlines their margins, as in Circle V of Fig. **54.** When this pigment disappears also it leaves a pure white surface, that of the sclera. In spite of the fact that the vessels can no longer be seen we are justified in ascribing these white spots to vascular changes if we can find sclerosed vessels in other parts of the fundus.

*4. The Form of the Change.*

The forms of some changes are so characteristic that we can deduce from them at once some conclusions as to their etiology. Among these are:

(*a*) Ruptures of the chorioid after contusions.

These are white, usually crescentic, and lie concentrically to the margin of the papilla. Their margins are sharply cut and frequently covered with pigment. In most cases they lie to the outer side of the papilla, more rarely to its nasal side. The retinal vessels pass smoothly over them.

In rare cases the ruptures are situated more peripherally, at the place where the blow was received, and are not concentric to the papilla. In such cases the lesion is to be considered as due not to contrecoup, but to the direct effect of the injury.

(*b*) Large, elevated, brilliant white patches, strewn richly with pigment, at the posterior pole of the eye, chorioretinitis proliferans, are indicative of transverse gunshot wounds of the orbit.

*5. Differences of Level.*

Finally the aid of parallactic displacement must be sought in the examination, because depressions and elevations from the ordinary level are met

with. First we have to think of proliferations of connective tissue, some of which are caused by injuries, some by general diseases.

Differences of level can also be detected in coloboma of the chorioid, of the macula, and of the optic nerve, in the so-called "staphyloma verum," and in tumors of the chorioid.

## B. Special Diagnosis

Note: As the retina is always secondarily involved in diseases of the chorioid the term chorioretinitis is preferable to chorioiditis.

## (a) Changes in the Chorioid and Retina Which Occur Chiefly or Exclusively in the Periphery

We will begin with these because they are the most common, and will consider first the diseases that are characterized by

## Collections of Pigment

1. Collections of pigment that resemble *bone corpuscles* are found in *chorioretinitis pigmentosa*, or, a better name for the disease, pigment atrophy of the retina. Fig. **51** shows these collections in their characteristic form. They are of a deep black color, may be more or less abundant, and may be rather lumpy, as depicted on the plate, or may be more delicate in their tracery. The form, which is suggestive of the microscopic appearance of bone corpuscles, is brought about by the fact that the pigment follows along the branching course of the smaller arteries and the capillaries of the retina. Not infrequently even the large arteries may be seen to have mantles of pigment. At first the pigment is only sparsely present, but it gradually becomes more abundant as the disease progresses toward the macula. The vessels of the retina are greatly contracted. The markings of the chorioid become visible in consequence of the atrophy of the pigment layer, and obliterated vessels can often be seen distinctly. The entire fundus acquires a peculiar, leaden color, from which the disease can frequently be recognized at the first glance. The papilla is atrophic in advanced cases, and usually has a yellowish hue (see page 52).

The central vision may remain good for a long time, but the functional disturbance in the periphery is shown by a contraction of the visual field and the onset of a marked hemeralopia.

A form of chorioretinitis is also to be met with which exhibits the same subjective symptoms as pigment atrophy, but is destitute of pigment. This is known as retinitis pigmentosa sine pigmento. Retinitis punctata albescens, which causes similar subjective symptoms, but is characterized by the presence of little white spots, has a similar etiology.

The disease is congenital and usually occurs in children whose parents

are nearly related. This fact differentiates it from chorioretinitis pigmentosa secundaria, which exhibits collections of pigment of a similar shape as the consequence of a plainly demonstrable sclerosis of the vessels of the chorioid, often confined to circumscribed portions of the periphery (see Fig. **52**), but the secondary pigmentations are usually of a less regular form (compare with Fig. **67**).

2. A second form of pigmentation of the periphery of the fundus is by *masses of pigment, which are often annular,*
as shown in Fig. **53**. This pigmentation is by no means as regular and is not as delicate as that seen in chorioretinitis pigmentosa; it is usually found only at certain places in the periphery. The retina and the optic nerve are not as seriously involved. This is one type, the grosser, of hereditary syphilis of the eye, the other is characterized by the so-called

3. *Snuff fundus,*

shown in Fig. **63**. The pigment is present here in the form of very minute points, between which are little, roundish depigmentations. These produce an appearance that has given rise to the name "pepper and salt fundus." Although this is fairly rare in a distinct form, cases in which it is suggested are rather common. The changes are to be seen most frequently in the lower periphery of the eye.

4. *Isolated spots of pigment,*

such as are seen in Fig. **80,** are likewise found in the periphery. They are usually surrounded by a bright halo. They are caused by either hereditary or acquired syphilis. When they are due to hereditary syphilis the former existence of an interstitial keratitis can frequently be proved either by the history, or by finding blood vessels situated deeply in the tissue of the cornea.

*Depigmentation in the Periphery.*

No attention will be paid here to such depigmentations as occur accidentally in the periphery, like those shown in Fig. **75,** but only to those that are typical.

1. *Discrete Depigmentations.*

These are met with in the pepper and salt fundus, in which the little round, bright spots may be more numerous than the dots of pigment. Such a fundus is shown in Fig. **55**. In many cases the spots may be mistaken for colloid deposits on the vitreous lamella, which are described on page 127, and depicted in Fig. **41,** but they are rather larger, and are to be found chiefly in the periphery. It is only in severe cases that they extend as far as the papilla. They are to be seen in childhood, while colloid deposits first appear in old age, as a rule. They are due to hereditary syphilis and are often met with in company with other signs of that disease, such as diseases

of the vessels and pigmentations. A very bad case of this nature is shown in Fig. **56,** in which the retina is involved through destruction of its vessels, and the optic nerve is atrophic.

2. *Superficial Depigmentations,*

as the result of the destruction of the choriocapillaris, are likewise often due to hereditary syphilis. The peripheral part of the fundus is brightened and the large vessels of the chorioid can be seen, although they are invisible in the central portion. This can be distinguished from a partial albinism, such as is shown in Fig. **8,** by the smallness of the arteries of the retina and the presence of detached spots of pigment.

These depigmentations, which are usually found only of the extent and intensity depicted in Fig. **57,** may spread more or less over the fundus in exceptional cases, but even then they are most marked in the periphery. Fig. **58** is a picture of such a case, in which the retina and optic nerve were involved as well as the chorioid.

## (b) Changes in the Chorioid in the Region of the Macula

The diffuse changes in the chorioid, which will be described later, may involve the region of the macula under certain conditions, as shown in Fig. **80,** but it is proposed to deal in this place only with the typical diseases of the macula that are characterized by certain peculiarities. It may be stated here that in the beginning of a disease of the macula the region of the latter appears to be slightly hazy, and that the reflex ring about it is abolished.

1. *Arteriosclerotic Changes in the Macula.*

This is often called senile degeneration, yet it is by no means confined to old age, for premature arteriosclerosis may occur in young persons occasionally, the same as senile cataract. The varying types and intensity of such changes are depicted in Figs. **59** to **62.** The markings of the chorioid stand out with unusual distinctness, perhaps with abnormal touches of pigment, or little bright, yellowish, or reddish yellow points may be seen with a more or less abundant proliferation of pigment between them. Colloid deposits are sometimes found in their vicinity. The changes are very trivial and cause no symptoms at all, unless they happen to be situated in the center of the macula. Aside from these relatively insignificant changes in the macula, which are not infrequently associated with a development of connective tissue, scleroses are to be found in the large vessels, as shown in Fig. **64.** Such an arteriosclerosis can be simulated, in rare cases, by a nephritic disease of the vessels of the chorioid, but it rarely happens, in such cases, that such lesions of the retina as œdema and hemorrhages are lacking (see the nasal side of the papilla in Fig. **69**).

**2 .** *Changes in the Macula Caused by Contusions, or by the Presence of a Foreign Body in the Eye*

resemble closely those that have just been described. For this reason, and because it is quite seldom that they are to be seen, it does not seem best to devote a picture to them.

**3.** *The Changes in the Macula Caused by High Myopia* have been thus described by *v. Michel:*

"Sometimes little bright spaces are found in the layer of pigment epithelium, and at the same time small heaps of pigment, or short bright stripes, arranged in rows or formed into a network. In many cases whitish bands or lines, not edged with pigment, extend out from the margins of the staphyloma, which are joined in the region of the macula by transverse lines (Fig. **71**), or yellowish points may be visible near one another. In other cases a hemorrhage, or a dirty gray, or greenish little elevation, or a deep black spot of pigment, takes the place of the macula, when hemorrhages may be present in the vicinity, and it sometimes happens that a thrombosed vein of the chorioid is to be seen, running near the entrance of the optic nerve and toward the macula. Aside from the possibility of an immediate extension of the staphyloma to the region of the macula in cases of high myopia, the place of the macula may be occupied by a single white, sharply defined large spot (Fig. **72**), or several smaller ones of the same nature, bordered by a more or less broad, often irregular, fringelike edge of pigment, frequently in the form of a ring."

The changes that take place on the papilla, as well as the shadows of the so-called staphyloma verum (Fig. **73**), have been mentioned on page 37.

These changes in myopia are to be ascribed to the effect of the stretching exerted upon the vessels of the chorioid, and upon the tissue of both this membrane and the retina, in the temporal portion of the posterior segment of the globe, which sometimes causes ruptures and apertures in the elastic lamina, as well as disease of the chorioidal vessels.

**4.** *The So-called Coloboma of the Macula.*

This is a roundish, or transversely oval spot in the region of the macula, which measures 3 P. D. horizontally by from 1 to 3 P. D. vertically, has an edge of pigment, and usually present a network of the same. Its comparatively large size and its regular margins differentiate this from other spots in the chorioid, with which it is frequently associated.

## (c) The Changes in the Chorioid about the Optic Nerve,

such as the conus, the staphyloma, the halo, and peripapillary atrophy of the vessels, have been dealt with for the most part on page 33.

## Peripapillary Sclerosis of the Vessels

may be mentioned again. Arteriosclerosis affects two regions, that of the macula, and that of the entrance of the optic nerve.

In Fig. **65** may be seen plainly sclerosed large vessels, some of them still containing slender columns of blood, which resemble those shown in the schematic drawing in Fig. **54**. This form of atrophy of the chorioid is commonly progressive, and may spread from this place over the entire fundus (Fig. **67**). More or less abundant, irregular heaps of pigment appear at the same time. The vessels of the retina may, or may not be involved in the sclerosis. The prognosis of sclerosis of the vessels of the chorioid is by no means as bad as that of sclerosis of those of the retina (see page 153).

Such an extensive vascular sclerosis may be caused by hereditary syphilis in exceptional cases. Fig. **68** portrays a case of this nature in which the vessels of the chorioid were sclerotic over almost the entire fundus. It is only in the region of the macula that a place can be seen which, although changes are present, is still capable of performing its functions.

## Ruptures of the Chorioid

are characteristically situated about the entrance of the optic nerve (see page 126 and Fig. **83**). This is true also of the typical

## *Coloboma of the Chorioid* (Figs. **84** and **85**).

Colobomata of the chorioid lie below the papilla, in the inverted image above, and have the form of an egg, or of a shield. The greater diameter is always vertical. The color is a brilliant white, with which a gray blue tone is often mixed, and sometimes some brownish places can be seen. A special tinting is given by irregular excavations which form little hills and valleys. In many cases the coloboma is bordered by a sharply defined, black edge of pigment, and flecks of pigment can often be seen in the coloboma itself. In addition to the vessels of the retina, which run smoothly over the white surface, branches of the ciliary vessels that pierce the sclera can usually be seen; these are often twisted like corkscrews. The size of the coloboma varies; in many cases it is situated quite peripherally and can be seen only when the patient looks far downward, in others it begins just below the papilla (Fig. **84**), and it may extend above the disk and involve the macula (Fig. **85**).

The extent of a coloboma does not necessarily accord with a corresponding defect in the field of vision; on the contrary, the latter may sometimes be perfectly normal; this depends on the extent to which the retina is involved.

A coloboma is to be considered as a true arrest of development, due to a faulty closure of the fetal ocular cleft. Frequently it is met with in com-

pany with other malformations of the eye, such as coloboma of the iris, microphthalmos, strabismus, and nystagmus, as well as of other parts of the body.

An ophthalmoscopic picture which is seldom seen is known as **coloboma of the optic nerve.** The papilla is doubled in size and appears as a roundish, or vertically oval hollow.

(d) *The Disseminated Form of Chorioretinitis* is characterized by the fact that it appears in multiple spots, little if any larger than the papilla, which are usually situated at the posterior pole of the eyeball. These spots may be fresh or old.

*Fresh spots* are small, roundish, rarely elongated, gray, and about a quarter of the size of the papilla when they are not confluent. The spots themselves cannot be seen, the accompanying œdema of the retina alone is visible. They can be perceived most readily when a vessel of the retina happens to pass over them (see the vein running downward and inward in Fig. **78**): the vessel is raised at such a place, is wavy, and in many cases is covered partly or wholly by the œdema.

These spots are usually caused by tuberculosis or syphilis. The latter disease is particularly to be suspected when they lie in the anterior segment of the chorioid, and when the diagnostic signs mentioned on page 170, deep vessels and central opacities of the cornea, dots of pigment on the lens, and sclerotic vessels of the chorioid, are present. But syphilitic diseases of the chorioid very often manifest themselves from the start through a sclerosis of its vessels (compare with the schematic drawings in Fig. **54**).

With regard to the differential diagnosis from spots having a similar appearance, see page 126.

*Old atrophic spots* are found in a great variety of forms. The development of a fresh spot into an atrophic one can be watched in comparatively few cases. We usually see the picture produced by the bygone process and have to try to draw conclusions from it. It is clear at once in most cases that the spot is chorioiditic and not retinitic, for a pigmentation characteristic of a chorioiditic spot is almost never lacking either in the spot itself, or in its immediate vicinity. Syphilitic spots are usually much more abundantly pigmented than the tuberculosis, so that a weak pigmentation is indicative of tuberculosis. The color may be yellowish, yellowish gray, or pure white: this depends on whether portions of the chorioid are present, or the membrane has been completely destroyed. The white color is brought about either by the sclera shining through the atrophic chorioid, or by the formation of a hyaline, cicatricial, connective tissue in the place of the membrane that has been destroyed.

Atrophic spots are divided into two varieties:

1. Those that are *without* visible changes in the vessels;

2. Those that are *with* visible changes in the vessels.

Tuberculosis is the principal cause in cases that belong to the first group,

while syphilis, arteriosclerosis, and nephritis must be taken into account in those that belong to the second.

*1. Atrophic Spots Without Visible Changes in the Vessels.*

One of the most important services rendered to the world by *v. Michel* was to call attention to tuberculosis as one of the main causes of chorioretinitis. Although this idea met at first with the strongest opposition, he had the satisfaction of seeing it adopted by almost all ophthalmologists. It was he also who pointed out the diagnostic importance of the changes in the vessels. Although we must generally leave the final determination of the etiology of a disseminated chorioiditis to the general examination, yet the differential points mentioned do good service.

The number of the spots is extremely variable; sometimes only a single one is accidentally discovered in an ophthalmoscopic examination, sometimes great numbers are scattered over the entire fundus. Fresh spots may be present along with old ones. The spots are usually roundish, frequently confluent, and have festooned margins. In their vicinity is often to be found a depigmentation of the pigment epithelium, which looks like the decoloration produced by a chemical substance. The pigmentation commonly is slight. Such a sharp pigment edge as is observed in syphilis (Fig. **74**) is almost never seen. The pigment usually lies in other places, near by, yet not immediately connected with the spots. At most the spot has in its center a little dot of pigment, as though the nodule in its growth had lifted up a little piece of the pigment epithelium.

The combination of such details may give rise to the greatest variety of pictures, as may be seen in Figs. **78** and **79**.

The vessels of the retina are intact and, in many cases, are rather fuller than usual.

*2. Atrophic Spots with Changes in the Vessels.*

The essential points regarding the appearance and origin of such diseases of the chorioid as are produced by changes in the vessels have been described with the aid of schematic drawings. When we look at Fig. **74** we find that the schematic drawing resembles actuality very closely. The number, size, and form of these spots vary a great deal. Usually, when the lesion does not spread diffusely, they are bordered by a distinct, black ring, and more or less abundant masses of pigment, which vary in size and number, are also present. In rare cases the spot is covered by a network of pigment that resembles in its form chorioretinitis pigmentosa (Fig. **52**).

Under certain circumstances a quite diffuse arrangement of changes in the vessels of the chorioid, heaps of pigment, and totally atrophic places is brought about by the confluence of separate spots, as is shown in Figs. **76** and **77**.

Syphilis, arteriosclerosis and nephritis are the chief causes in these cases, less often they are due to diabetes, or malaria. Hence a general examination is always necessary.

The degree to which the vision is disturbed depends mainly on the involvement of the macula; at first objects seem to be distorted, i.e., the patients have metamorphopsia, later central relative and absolute scotomata appear. Sometimes we are surprised to find that the vision is comparatively good in spite of the extensive changes in the fundus.

*Changes in the Chorioid*
*with Differences of Level.*

The ordinary changes in the chorioid generally cause no differences of level, as a defect that may be produced is balanced by a compensating proliferation of tissue, and the reverse. Yet in a number of these diseases a difference of level can be detected by means of parallactic displacement and the observation of bends in the course of a vessel, showing either a hollow or an elevation.

1. A *hollow* is found chiefly in colobomata of the chorioid (see Fig. **85**), and in many cases of high myopia. In the latter it is known as a staphyloma verum (Fig. **73**).

2. An *elevation* is observed in tumors, either inflammatory or noninflammatory, and in detachment of the chorioid.

The *noninflammatory tumors*, such as sarcoma and metastatic carcinoma of the chorioid, make themselves manifest through the accompanying detachment of the retina and have been described in connection with this condition, see page 152.

*Inflammatory tumors* are of either a syphilitic, or a tuberculous nature. The former, gummata, are accustomed to start with very severe signs of inflammation, which extend far into the neighboring tissues, and often render an exact diagnosis impossible by an opacity in the vitreous. After they have cleared up under suitable treatment only a harmless atrophic spot is usually to be seen at the affected place.

*Tuberculous inflammation*, conglomerate tubercle, may greatly resemble a gumma at first, but its result is commonly not so benign. It often perforates the sclera and proliferates into the tissues of the orbit, rendering exenteration of that cavity necessary. The conglobate tubercle near the head of the optic nerve may threaten life through the production of a meningitis by proliferation along the sheath of the optic nerve.

In benign cases the nodules are transformed into connective tissue, which gives the impression of a mountain covered with snow by its bright color and its elevations and depressions. Very little pigment is found in its neighborhood.

*Detachment of the chorioid*, both circumscribed and total, occurs, but it is rarely diagnosed during life, it is observed more often in pathological preparations.

It appears as a dark surface with parallactic displacement of the vessels of the retina, but perceptible markings of the chorioid (compare with detachment of the retina, page 152). When it is particularly large it appears as a light brown protrusion.

It is observed most often with hemorrhages into the perichorioidal space, such as occur in high myopia and after wounds, in congenital buphthalmos, and after extraction of cataract. Total detachment takes place in phthisis bulbi.

Elevations due to the *proliferation of connective tissue* in the chorioid, chorioretinitis proliferans, such as are observed in arteriosclerosis, tuberculosis, and especially after wounds, are relatively frequent. Chorioretinitis proliferans forms a typical picture after transverse shot wounds of the orbit, with the production of white, elevated spots associated with an extremely rich development of pigment. The way in which this is brought about is that the eyeball is pressed in from behind by the explosive force of the shot passing rapidly through the orbit, and the chorioid is thereby ruptured in many places. The excessive cicatrization set up in healing, together with the organization of the hemorrhage, gives the characteristic picture.

PLATE XXVIII

Fig. 51.  Retinitis Pigmentosa, or Pigment Degeneration of the Retina

Fig. 52.  Secondary Retinitis Pigmentosa

## Fig. 51.   Retinitis Pigmentosa, or Pigment Degeneration of the Retina

(See page 174)

The tyro is apt to call every disease of the fundus associated with collections of pigment retinitis pigmentosa, but this name should be applied only to a certain distinctive clinical picture, with the exception of secondary retinitis pigmentosa, described below. *The form and position of the spots of pigment* are characteristic. They lie in the outermost periphery and advance gradually toward the papilla. They resemble in form the microscopic picture of bone corpuscles, because the pigmentation follows the capillaries of the retina. It also follows the larger vessels, which sometimes have mantles of pigment (see also Fig. **57**). Other characteristics are the color of the papilla and the color of the fundus. The papilla is of a waxy yellow in marked cases, and the fundus has a blue gray tone, which may suggest the correct diagnosis at once. The vessels of the retina are quite drawn out and of very small caliber. In most cases an involvement of the chorioid is shown by the presence of sclerosed vessels and depigmented places. Such a condition may be seen in this picture in the vicinity of the papilla.

Among the subjective symptoms are an extreme concentric contraction of the field of vision, and a functional disturbance, the so-called hemeralopia. The predisposition to the disease is congenital; it often affects children of consanguineous parents.


## Fig. 52.   Secondary Retinitis Pigmentosa

That form of retinitis pigmentosa in which the disease of the vessels of the chorioid is particularly marked is designated by this term. Changes in these vessels are met with in the typical form of the disease, as was mentioned above, so this one differs from it only in degree.

But it is customary also to call a condition "secondary retinitis pigmentosa" which in no way corresponds in etiology, extent, or state of the papilla, to the typical form, although the shape of the deposits of pigment is the same. Such a condition is shown in this picture, which was taken from the eye of a woman 50 years old, who had arteriosclerosis. The changed vessels of the chorioid, which have produced an atrophy that resembles a staphyloma, can be seen distinctly in the vicinity of the papilla; similarly sclerosed vessels may also be seen in an area that lies far in the periphery. This area is covered by a network of "bone corpuscles." A similar network can also be seen at a place where the sclerosed vessels are not so plainly visible.

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Tab. **28.**
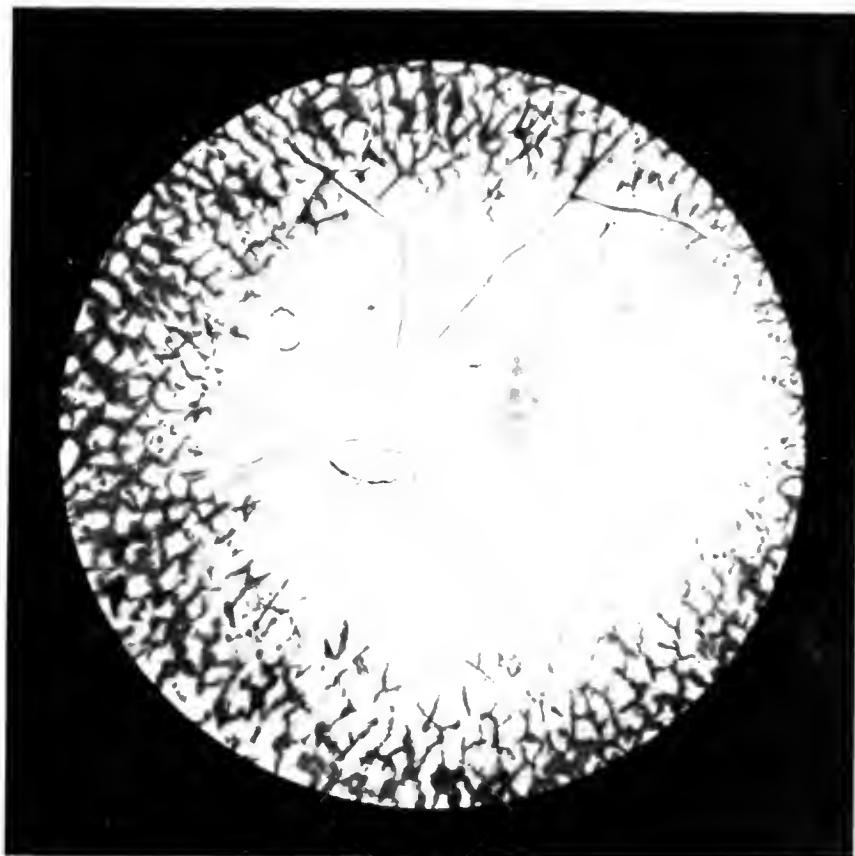


Fig. 51.
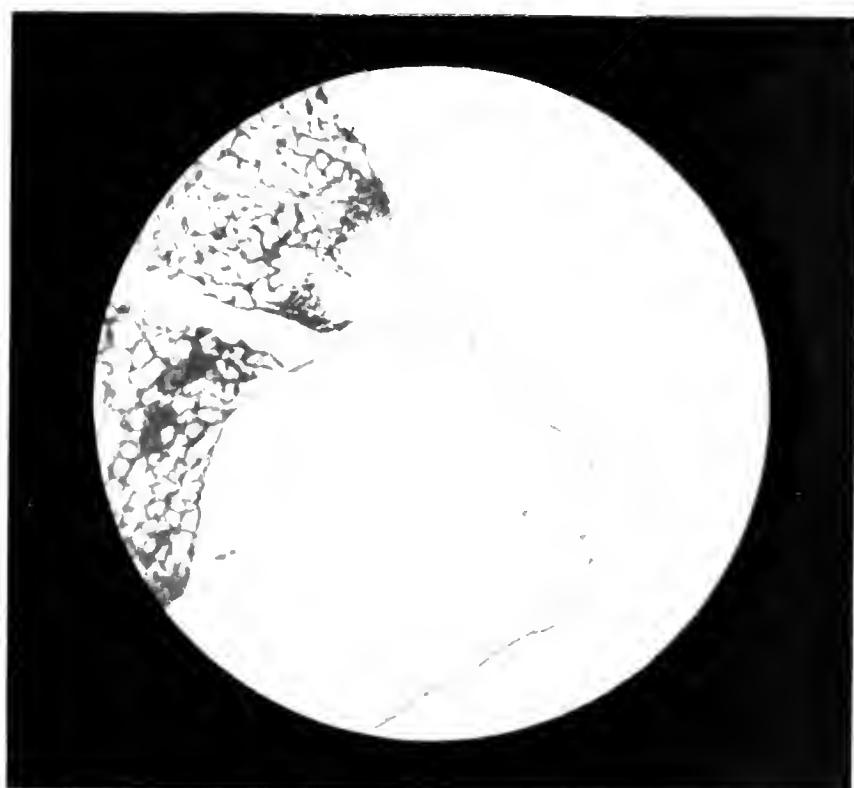


Fig. 52.

Fig. 53.  Grossly Pigmented Fundus of Hereditary Syphilis

## Fig. 53. Grossly Pigmented Fundus of Hereditary Syphilis

In contrast with the preceding pictures the pigment is seen in this one to be in large, round masses, which have a distinctly circular form in the places where it is less dense; in other places the pigment has blended into an inextricable network of black points. The detached groups of spots are separated by a zone of depigmented tissue from the normal. A depigmented zone is also to be seen about the papilla.

The papilla and the vessels of the retina are normal.

This is a condition that is met with rather frequently in eyes that have suffered from interstitial keratitis. It is sometimes to be seen throughout the entire periphery, sometimes to be found only in separate circumscribed areas.

Tab. **29.**
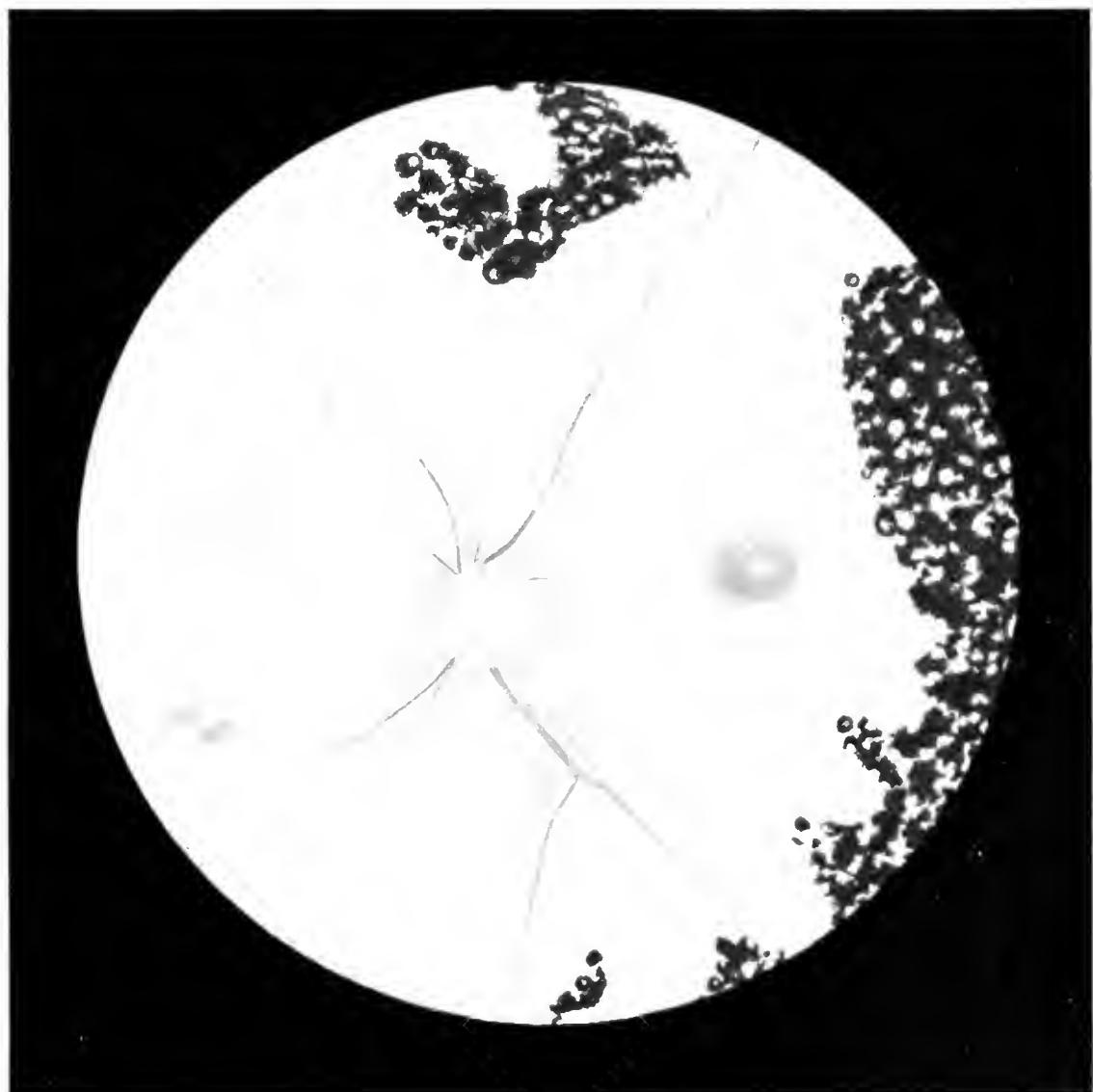


Fig. 53.

PLATE XXX

Fig. 54.  Schematic Pictures of Diseases of the Chorioidal Vessels

**Fig. 54. Schematic Pictures of Diseases of the Chorioidal Vessels**

(For the explanation of this plate see page 168)

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Tab. **30.**



Fig. 54.

} Sclera
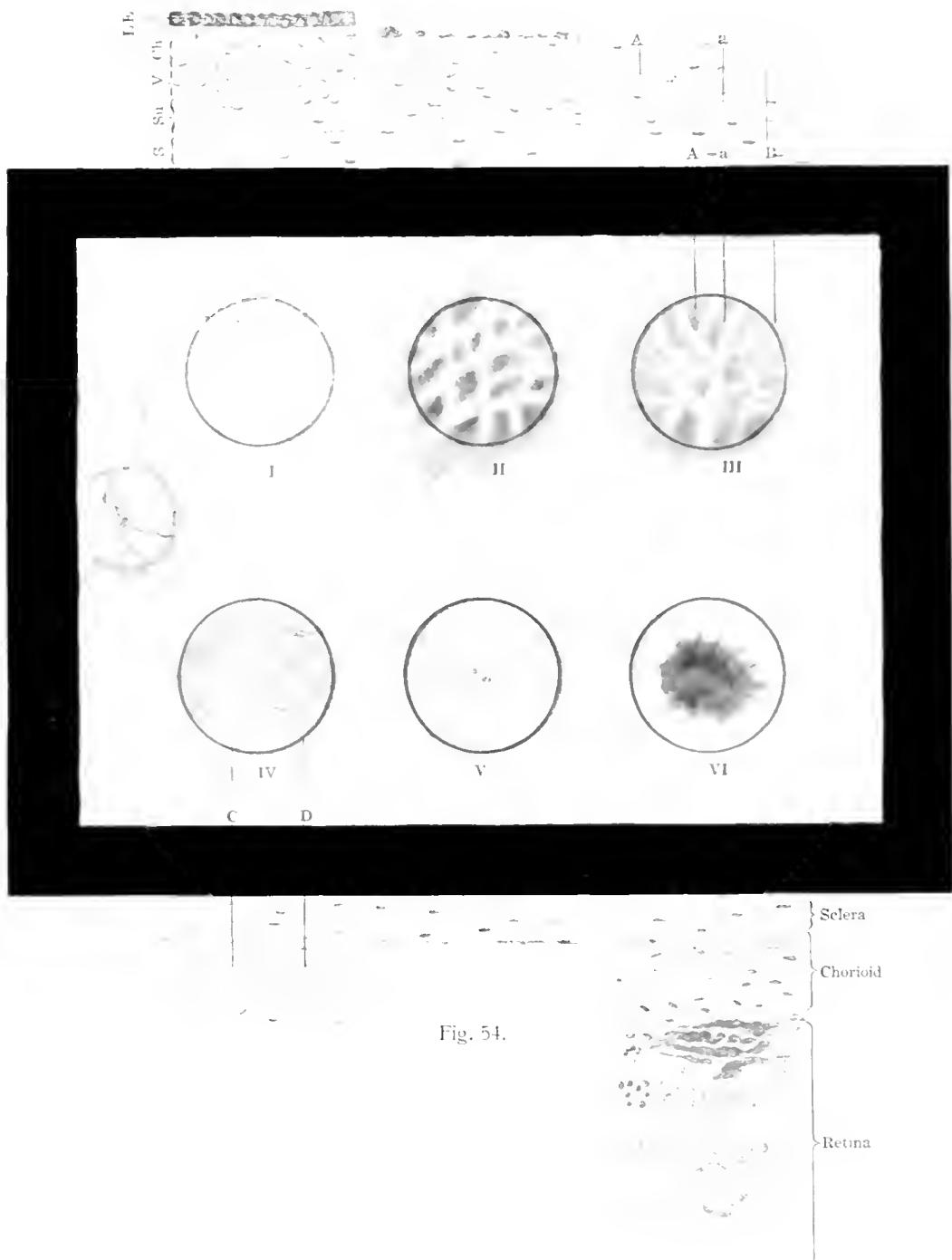
} Chorioid

} Retina

PLATE XXXI

Fig. 55.  The So-called Pepper and Salt Fundus of Hereditary
Syphilis

Fig. 56.  Very Severe Chorioretinitis Due to Hereditary Syphilis,
with Atrophy of the Optic Nerve

## Fig. 55. The So-called Pepper and Salt Fundus of Hereditary Syphilis

(See page 175)

The papilla and the vessels of the retina are normal. The density of the pigment epithelium varies in different parts of the fundus. In some places it hides the chorioid completely, in others the markings of the latter are plainly visible. A patch of distinctly sclerosed vessels is to be seen in the vicinity of the optic nerve. The characteristic feature in this fundus is the presence of numerous little, roundish depigmentations, which are often surrounded by halos of denser pigment.

## Fig. 56. Very Severe Chorioretinitis Due to Hereditary Syphilis, with Atrophy of the Optic Nerve

This picture was taken from the eye of a boy 13 years old.

The most striking feature is the almost total absence of retinal vessels, of which only very small, sclerosed traces can be seen in the immediate vicinity of the papilla.

The papilla itself is perfectly white. On account of the total absence of an excavation and the invisibility of the meshes of the lamina cribrosa, the atrophy must be supposed to be of inflammatory origin in spite of the fairly sharp margins. Sharp margins are rather frequently found in cases of neuritic atrophy that develop during childhood.

Near the papilla can be seen some sclerosed vessels of the chorioid. The rest of the fundus is of a dirty gray color, in which few details are visible, with the exception of a few dots of pigment and some bright spots that faintly remind one of the depigmentations seen in the preceding picture. Some of the vessels of the chorioid are also visible in the uppermost part.

This boy had suffered from an attack of syphilitic meningitis in early childhood, which caused an optic neuritis with a subsequent atrophy, and at the same time the serious disease of the vessels of the retina and chorioid asserted itself.

The vision of this eye naturally was nil.

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Tab. **31.**



Fig. 55.
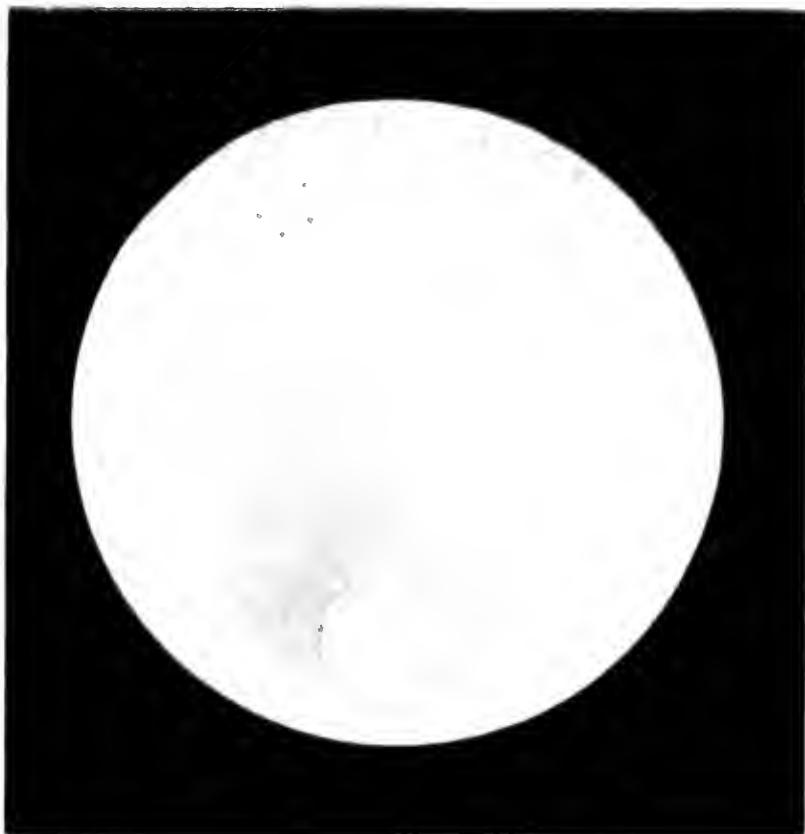


Fig. 56.

PLATE XXXII

Fig. 57. Chorioretinitis Due to Hereditary Syphilis

## Fig. 57.  Chorioretinitis Due to Hereditary Syphilis

This picture presents another type of hereditary syphilis of the eye.

The papilla is rather paler than normal, especially in its temporal half. The arteries of the retina are very small.  Some of the veins of the retina have well marked mantles of pigment in their peripheral portions.

Only a few spots of pigment surrounded by bright areolæ can be seen in the periphery.

The markings of the chorioid can be seen very plainly in the periphery. This is because the pigment epithelium and the choriocapillaris have been destroyed (see page 176).

Tab. **32.**



Fig. 57.

Fig. 58.  Chorioretinitis Due to Hereditary Syphilis, with Atrophy
of the Optic Nerve

## Fig. 58. Chorioretinitis Due to Hereditary Syphilis, with Atrophy of the Optic Nerve

As in the case depicted in Fig. **56,** the condition here presented was preceded by a meningitis, a consequence of which was the atrophy of the papilla, which is surrounded by a very distinct ring of glia tissue.

The vessels of the retina are very small and drawn out.

The entire fundus shows a high degree of depigmentation, with the remains of the pigment grouped about separate roundish foci, which call to mind Fig. **55.**

It cannot be determined with certainty whether the crescentic reddish spot in the macula is a hemorrhage, or a deposit of pigment, because many of the small spots of pigment exhibit a reddish tone of color.

Tab. **33.**



Fig. 58.

## PLATE XXXIV

Fig. 59. Early Stage of Arteriosclerosis of the Vessels of the Chorioid in the Region of the Macula

Fig. 60. Senile Degeneration of the Macula

Fig. 61. Senile Degeneration of the Macula

Fig. 62. Senile Degeneration of the Macula

## Fig. 59.  Early Stage of Arteriosclerosis of the Vessels of the Chorioid in the Region of the Macula

(See page 176)

In this picture, taken from the eye of a man 60 years old, some of the vessels of the chorioid can be seen plainly in the macula. The fundus is otherwise normal. The place is surrounded by a circle of very minute bright spots.

The explanation of this picture is that a portion of the pigment epithelium has been caused to atrophy by a disease of the choriocapillaris, and that consequently the normally colored large vessels of the chorioid have become visible.

The white spots are due to colloid deposits on the vitreous lamella.

The vision in this case was $\frac{1}{8}$ of the normal.

## Fig. 60.  Senile Degeneration of the Macula

(See page 176)

In this case also some large vessels of the chorioid are visible, with a number of fine points of pigment near them; otherwise the fundus is normal. Vision was reduced to $\frac{1}{8}$.

The pigment epithelium of the retina and the choriocapillaris must have been destroyed, for otherwise the markings of the chorioid could not be seen.

## Fig. 61.  Senile Degeneration of the Macula

(See page 176)

Another form of degeneration in the macula is shown in this picture. A number of very minute, bright points are to be seen lying in a bed of pigment granules. Some areas of depigmentation are visible in the periphery. The papilla is too red in the picture, and the conus is too white, it should in fact be rather yellow. The spot of pigment is a portion of the pigment ring and denotes nothing pathological.

## Fig. 62.  Senile Degeneration of the Macula

(See page 176)

The changes in this picture are similar to, but grosser than those seen in Fig. 61. The papilla is surrounded by a white band that is too broad to be a physiological connective tissue ring. It must be supposed to be a senile halo, although its color is not quite right for this condition. The pigmented edge is likewise broader than the physiological pigment ring, and must be considered to be pathological.

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Tab. 31.



Fig. 59

Fig. 60
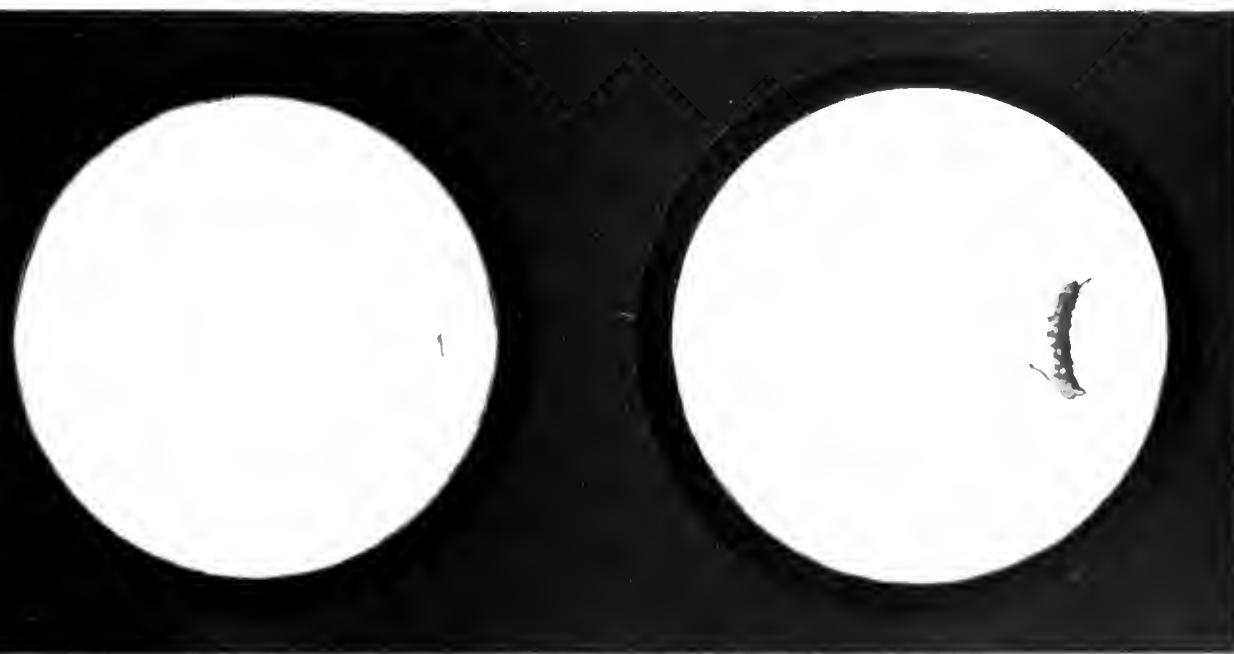


Fig. 61.

Fig. 62.

## Fig. 63. Finely Pigmented Fundus of Hereditary Syphilis, the So-called Snuff Fundus

(See page 175)

This is the snuff fundus, a finely pigmented type which is met with in hereditary syphilis, in addition to the grossly pigmented one shown in Fig. **53.**

Such a typical and distinctive picture as the one depicted here is seen comparatively rarely, but a less pronounced form is met with very often.

The papilla and retina are intact, the chorioid alone presents lesions, which are seen with the microscope to be a disease of the choriocapillaris with secondary disturbances of the pigment, and manifest themselves ophthalmo-scopically as finely granular heaps of pigment and round foci of degeneration.

## Fig. 64. Sclerosis of the Vessels of the Chorioid in the Region of the Macula

(See page 178)

This could also be called a senile degeneration of the macula if we were not accustomed to designate by this term such insignificant changes as those depicted in Figs. **59** to **62.**

The fundus is of the tessellated type, so that the vessels of the chorioid, with the deposits of pigment between them, are plainly visible in consequence of the thinness of the pigment epithelium. The vessels of the chorioid in the region of the macula are sclerosed, i.e., their walls have become thickened. The thickening of the walls is so great in places that a column of blood is no longer visible in some of the vessels, while only a slender column can be seen in the center of others.

The papilla is normal, the retinal arteries are rather small.

The reduction of vision is naturally very great in such cases; in this case the patient could only count fingers at 2 meters.
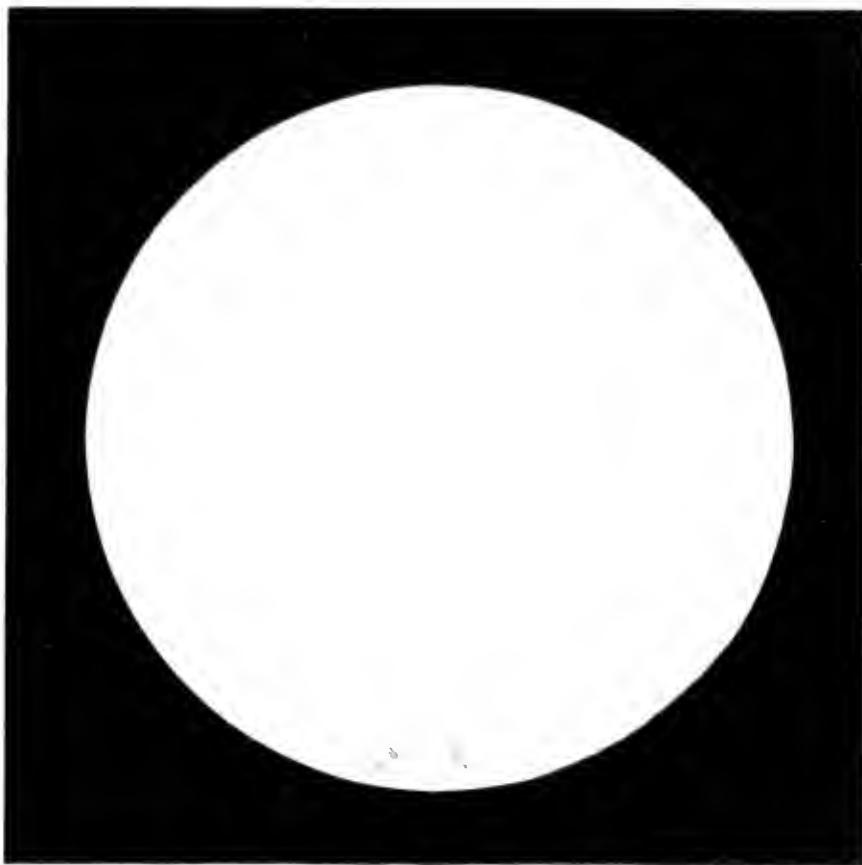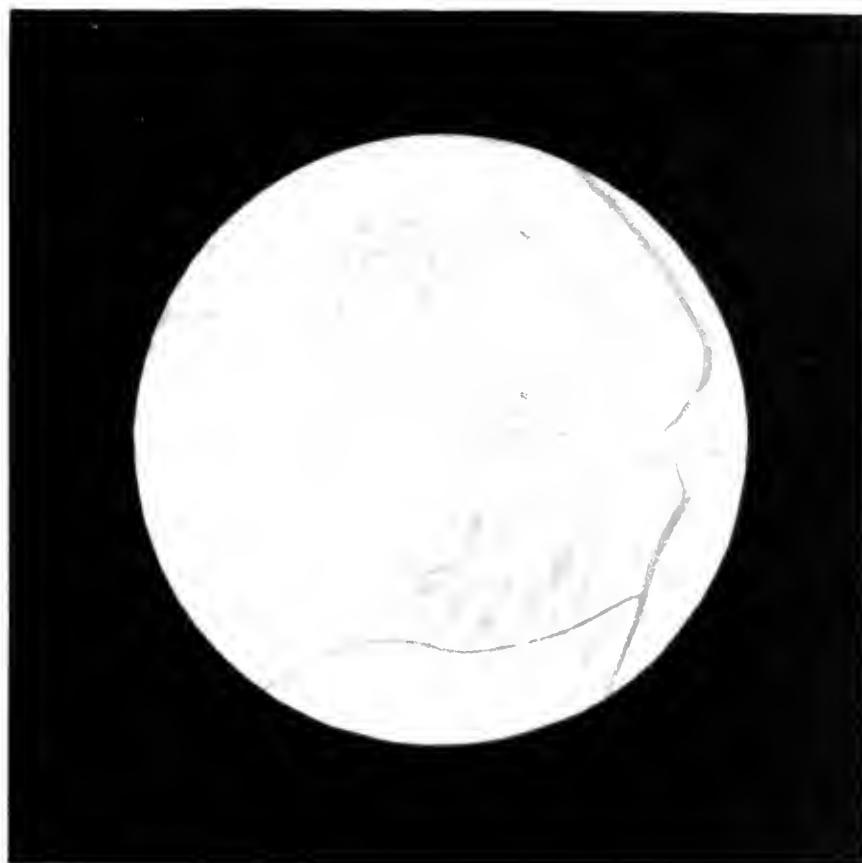
198

Tab. **35.**



Fig. 63.



Fig. 64.

## Fig. 65. Peripapillary Sclerosis of the Vessels of the Chorioid

(See pages 39 and 178)

The vicinity of the papilla, as well as that of the macula, is a favorite place for sclerosis to attack the vessels of the chorioid. In several places the vessels may be seen to be wholly obliterated, while in others they are only partly filled with blood.

## Fig. 66. Peripheral Patch of Sclerosis of the Vessels of the Chorioid

This might be mistaken at first glance for a coloboma of the chorioid if the intervascular spaces could not be seen so plainly. The vessels themselves are wholly obliterated and invisible. The reason why they still seem to be present is that the pigment which is normally situated between them still remains visible after they have disappeared, and outline the empty spaces left by them (compare with Plate XXX). If this pigment also should disappear a uniform white surface would be left. The lumps of pigment on the margin are due to proliferations of pigment. The presence of newly formed vessels renders it very probable that this condition was one of inflammatory origin (see page 104).

Tab. **36.**
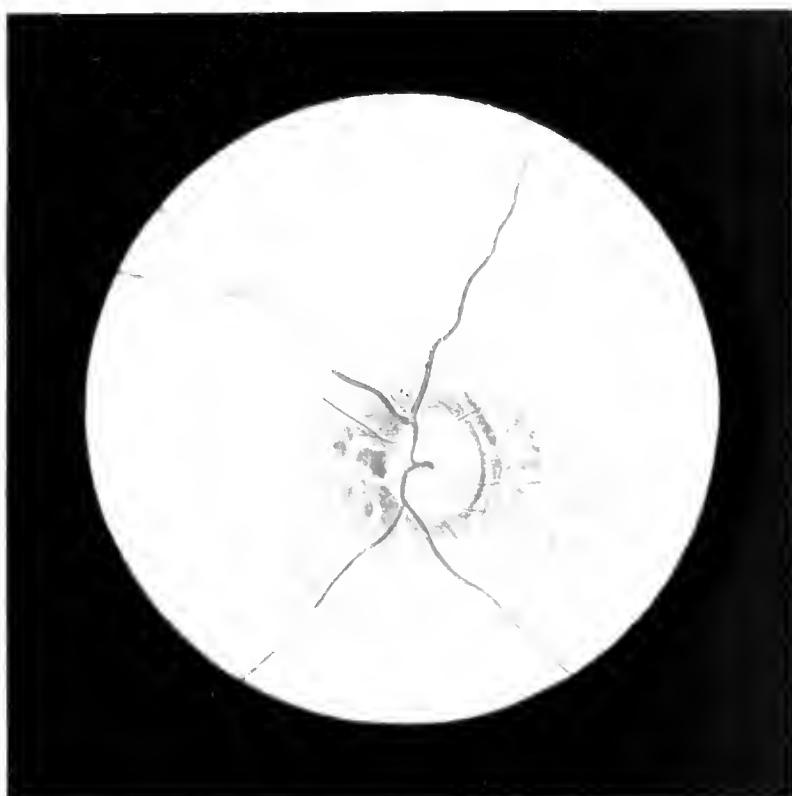


Fig. 65.



Fig. 66.

## Fig. 67.   Great Sclerosis of the Vessels of the Chorioid, and Less of Those of the Retina

(See page 180)

All of the vessels of the chorioid that are visible are sclerosed, and only a few of them contain slender columns of blood. The pigment epithelium must have been destroyed very extensively; the heaping up of pigment in various places is in harmony with such a destruction. The papilla is rather paler than normal (see page 55), and the vessels of the retina, particularly the arteries, are evidently contracted (see page 98).

## Fig. 68.   Extreme Sclerosis of the Vessels of the Chorioid

(See page 176)

The sclerosis is still more extensive in this case, and has reached a much greater intensity. While the intervascular pigment of the chorioid was preserved throughout its normal extent in the preceding case, in this one it is so atrophic that the time seems to be not far distant when the entire fundus will be transformed into a white surface. The region of the macula shows a trace of pigment epithelium, yet this also is changed. The papilla and the vessels of the retina are normal.

This vision was comparatively good, $\frac{1}{6}$ of the normal, but the visual field was concentrically contracted to an extremely small trace.

This case was met with in a girl 13 years of age who had hereditary syphilis. Her sister had a similar condition.

Tab. **37.**
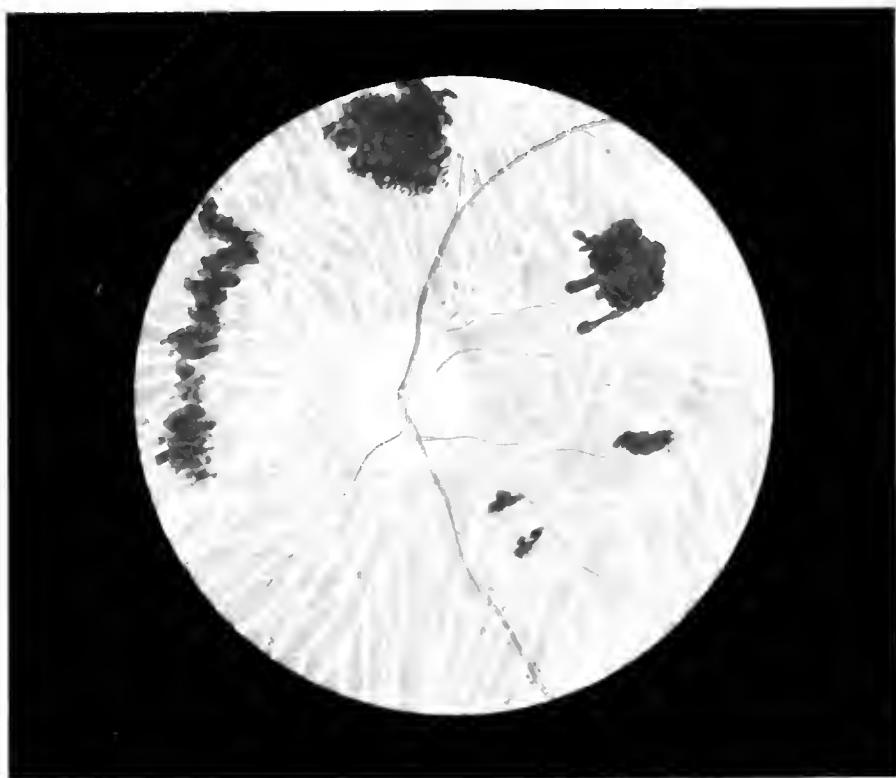


Fig. 67.



Fig. 68.

PLATE XXXVIII

Fig. 69.  Chorioretinitis Albuminurica

## Fig. 69.  Chorioretinitis Albuminurica

A picture similar to the preceding one may be produced by renal disease. In such cases, which are pretty rare, the signs of inflammation on the papilla and in the retina can scarcely be missed.  In the present case the nasal side of the papilla is distinctly hazy.  The retina shows plain signs of œdema by the haziness of its vessels, and contains some hemorrhages.

The vision is greatly impaired in such a case; in this patient it was reduced to counting fingers at 2 meters.

Tab. **38.**
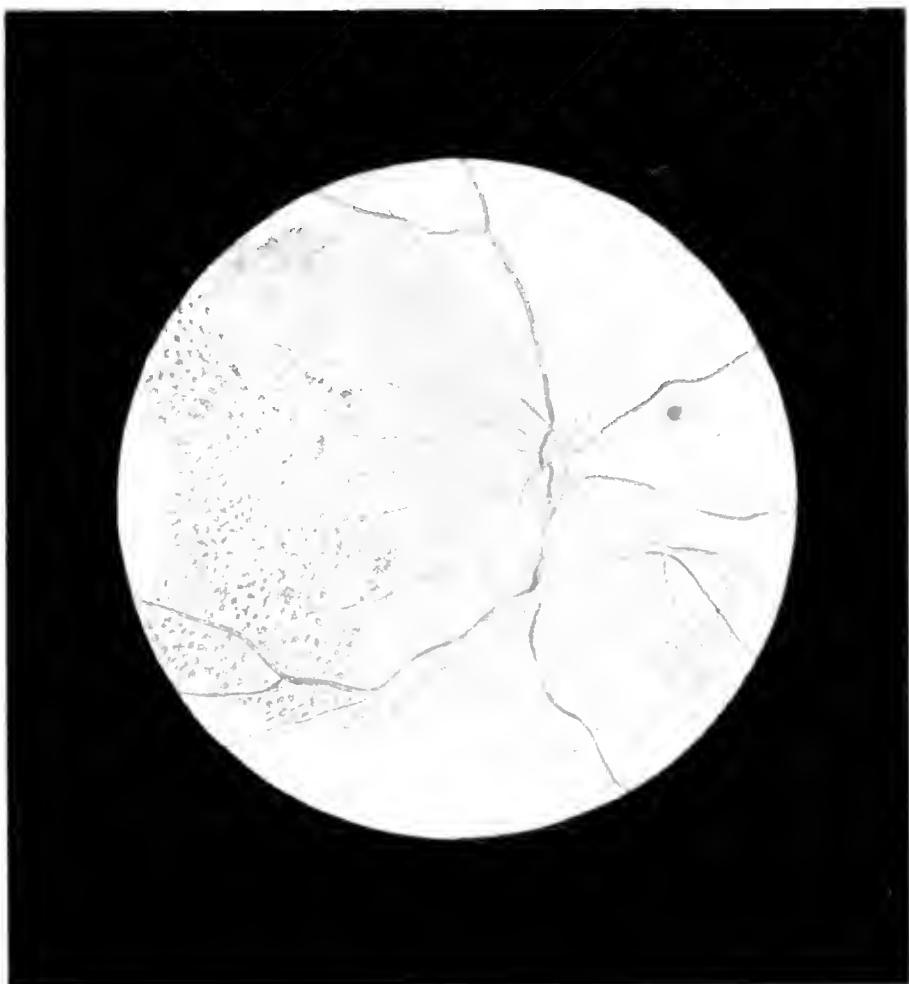


Fig. 69.

PLATE XXXIX

Fig. 70.  High Myopia; Temporal Staphyloma; Change in the
Macula

Fig. 71.  High Myopia; Circular Staphyloma; Sclerosed Vessels
of the Chorioid

## Fig. 70. High Myopia; Temporal Staphyloma; Change in the Macula

(See pages 37 and 177)

The papilla, which is vertically oval in this case, has a strikingly indistinct temporal margin; it seems to blend at this place with the staphyloma, which is situated altogether on its temporal side. See page 38 for the relations between the form of the papilla and that of the staphyloma. The latter is divided into 2 portions, one situated near the papilla, in which punctate markings can be seen, the other farther away, which is pure white. The punctate markings are due to traces of chorioidal pigment that have been left after destruction of the vessels of the chorioid (see page 37).

The vessels of the chorioid can be seen plainly in this very pale fundus, as well as some chorioidal hemorrhages to the nasal side of the papilla. In the region of the macula is a maze of white cords, which are explained by some as sclerosed vessels of the chorioid, by others as fissures in the pigment epithelium (see page 37).

## Fig. 71. High Myopia; Circular Staphyloma; Sclerosed Vessels of the Chorioid

(See pages 37 and 177)

This picture presents all of the characteristics of a myopic eye: staphyloma, pale fundus, stretched vessels of the retina, sclerosed ones of the chorioid. The papilla is normal, and is surrounded by a circular staphyloma, in the nasal side of which some traces of chorioidal pigment and one normal chorioidal vessel can be seen; all the other details have been destroyed. The rest of the chorioidal vessels can be seen very well in the pale fundus. The region of the macula, which is rather richly pigmented, shows a peculiar maze of white cords with little processes that remind one of the frosted branches of a tree. Some dots of pigment and some rather superficial depigmentations are also to be seen. It cannot be told with certainty whether all of the white cords are sclerosed vessels of the chorioid, or a part of them are due to fissures in the pigment epithelium (see page 37).

The myopia in this case was 12 diopters.

Tab. **39.**
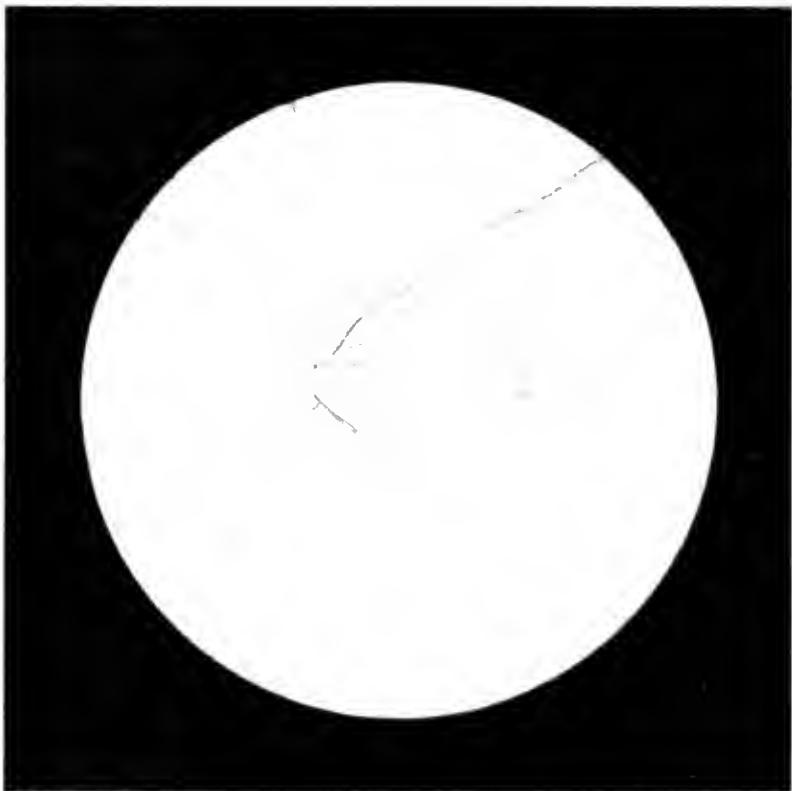


Fig. 70.
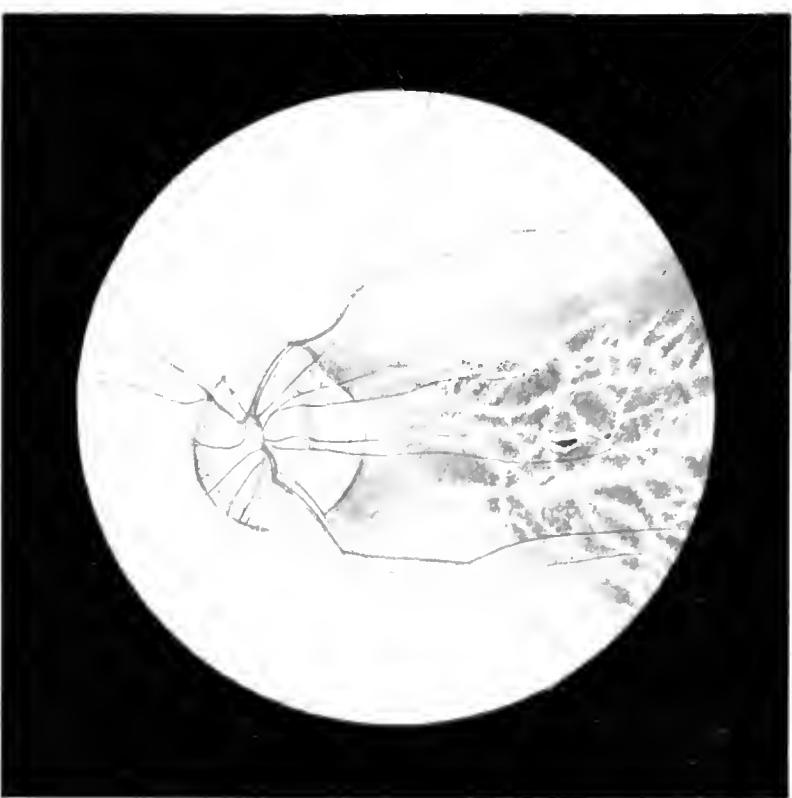


Fig. 71.

## Fig. 72. High Myopia with Circular Staphyloma and a Very Great Change in the Macula

(See pages 37 and 182)

The fundus is very pale, and some of the vessels of the chorioid can be seen quite plainly, as often happens in myopia. This is because of the stretching of the layer of pigment epithelium. The vessels of the retina are extremely drawn out and are smaller than normal. The papilla is surrounded by a circular staphyloma which is broadest upward and outward. A very careful examination reveals some remains of chorioidal pigment, which is the last trace of the chorioid that has undergone atrophy. The staphyloma is surrounded by a more or less strongly pigmented zone. Some small atrophic spots may be seen to its nasal side. The region of the macula is occupied by a large, kidney-shaped spot, measuring $2 \times 2\frac{1}{2}$ papillary diameters, in the hilus of which is a spot as large as the papilla, composed of numerous dots of pigment.

Above and below the latter lie several smaller spots of pigment, which are to be distinguished from the remaining chorioidal pigment by the intensity of their color. Sclerosed vessels of the chorioid, intermixed with white and black spots, lie to both the temporal and the nasal sides of this principal spot. The vision in this case was counting fingers at 2 meters with the correcting glass, — 16 D sph. ⊃ —2 D cyl.

## Fig. 73. High Myopia with a So-called Staphyloma Verum

(See page 26)

The papilla appears to be remarkably small, an optical illusion caused by the high degree of myopia (see page 17). It is surrounded by a circular staphyloma, which indicates that the vicinity of the papilla is pouched out rather uniformly (see page 38 concerning the form of the staphyloma). Parallel to the nasal margin of the papilla are to be seen one large and two small gray, or reddish gray, curved lines, in the region of which the vessels of the retina plainly bend. They are shadows cast by the margins of the protrusion of the posterior pole, sclerectasia, and consequently this has been termed staphyloma verum. Throughout its area the fundus is considerably brighter than it is elsewhere, because of the great stretching of the pigment epithelium. The vessels are extremely small, partly because of the stretching, partly because of an optical illusion. The arteries are drawn out, the veins are very tortuous, an unusual symptom in myopia, which may perhaps indicate a threatened detachment of the retina.

Part of the vessels of the chorioid are very clearly visible and some converge toward the papilla (compare with Fig. **3**). In the region of the macula these vessels are distinctly sclerosed in places, a preliminary stage of a greater change in the macula, such as may be seen, for example, in Fig. **71.**

The myopia in this case was approximately 35 diopters.

Tab. 10.



Fig. 72.
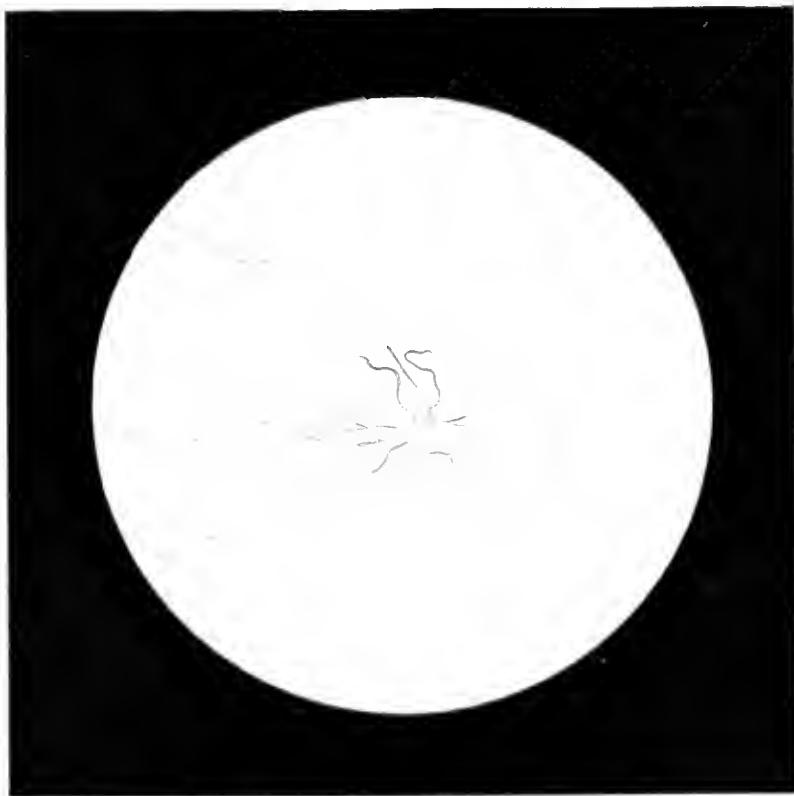


Fig. 73.

# PLATE XLI

Fig. 74.  Atrophic Spots in the Chorioid with Plainly Sclerosed
Vessels

Fig. 75.  Neuritic Atrophy of the Optic Nerve; Atrophic Spot in
the Periphery

# Fig. 74. Atrophic Spots in the Chorioid with Plainly Sclerosed Vessels

(See page 180)

Two sharply circumscribed spots of atrophy are to be seen in an otherwise normal fundus, one in, the other below the macula. The lower one resembles very closely one of the schematic drawings on Plate XXX. Sclerosed chorioidal vessels are to be seen with some pigment between them, but the latter has begun to disappear, so it is to be expected that the spot will be perfectly white within a short time, as the sclera will then be laid bare. Two of the vessels still contain blood, but the others are completely sclerosed. The spot is bordered by a ring of pigment.

The upper spot, which is due to the same cause as the lower, exhibits an abundant development of pigment, which forms a mass so shaped as to divide the spot into 4 smaller ones. The pigment epithelium has begun to become lighter in its neighborhood.

As soon as sclerosed vessels are noticed in the fundus we have to think chiefly of arteriosclerosis, syphilis, or nephritis as the cause, in the absence of myopia. Syphilis was the cause in this case.

# Fig. 75. Neuritic Atrophy of the Optic Nerve; Atrophic Spot in the Periphery

The atrophy of the papilla is shown to be neuritic by the indistinct margins and the great haziness of its surroundings. The latter indicates that the retina was involved to quite a considerable degree, and that the case was a very severe one of neuroretinitis. This corresponds to the actual conditions, for a choked disk preceded the atrophy and was caused by a gumma of the orbital portion of the optic nerve. The absence of any sheathing of the retinal vessels is noticeable. The pigment epithelium is denser in the upper part of the fundus than in the lower, so that the markings of the chorioid are not as clearly visible above, but otherwise the rest of the fundus is normal, except for a peculiar change to be seen in the extreme periphery, where a whitish spot with sharp outlines stands out amid normal surroundings. It is formed from more or less sclerosed vessels of the chorioid, between which traces of the pigment can still be seen. It is bordered by a beautiful ring of pigment. See Plate XXX for the pathology. The lesion in this case was due to a quite circumscribed syphilitic disease of the vessels.
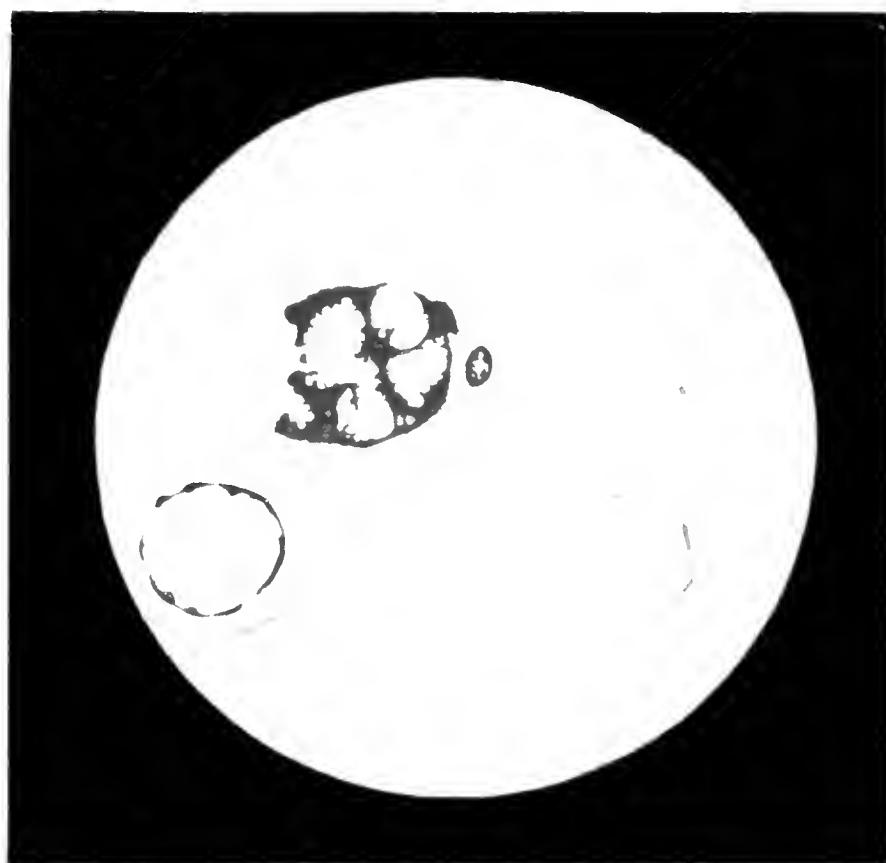
Fig. 74.
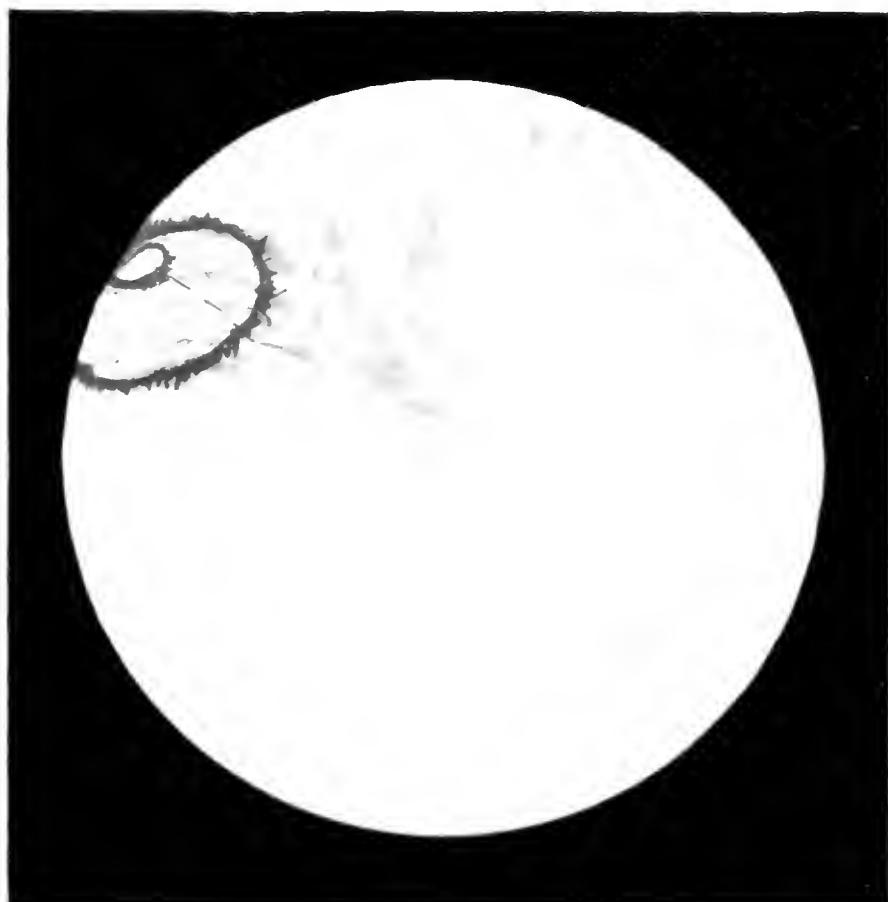


Fig. 75.

## PLATE XLII

Fig. **76.**  Extensive So-called Chorioretinitis Disseminata with Scleroses of the Vessels of the Chorioid

Fig. **77.**  So-called Chorioretinitis Disseminata with Scleroses of the Vessels of the Chorioid

## Fig. 76. Extensive So-called Chorioretinitis Disseminata with Scleroses of the Vessels of the Chorioid

The fundus shows serious changes throughout its entire extent, so far as it is depicted here, partly by patches of decoloration, partly by heaps of pigment.

The papilla and the vessels of the retina are normal; the latter pass smoothly over the lesions, hence the inner layers of the retina are likewise normal, and the lesions must lie in the outer layers of the retina and in the chorioid; they are changes in the pigment epithelium and scleroses of the vessels.

The larger part of the fundus to the nasal side of the papilla approaches the normal, yet, even in this part, lumps of pigment and sheathings of the vessels can be seen. Not a normal vessel of the chorioid is visible any longer in the rest of the fundus; above there are some vessels that are filled with blood, but even these are sheathed. Where the pigment epithelium between them has already been lost pure white places are to be seen, produced by the sclera covered with remnants of tissue. A disappearance of the pigment epithelium of the retina is the first requirement in order that these details of the sclera may become visible, but when the pigment leaves it settles elsewhere and gives rise to pictures that resemble this one. Of the three causes of disseminated chorioretinitis with vascular sclerosis, syphilis, arteriosclerosis and nephritis, the first was the agent in this case.

## Fig. 77. So-called Chorioretinitis Disseminata with Scleroses of the Vessels of the Chorioid

This picture exhibits changes that are quite similar to those shown in the preceding one, so much so that a separate description is not needed (see page 182).

The cause in this case was a nephritis gravidarum. The symptoms appeared chiefly in one eye during the first pregnancy and became much worse during the second. As the second eye was seriously affected in a third pregnancy premature labor was induced, after which both the albuminuria and the changes in the eye retrogressed. When the patient became pregnant a fourth time an abortion was induced at once.

Tab. 42.
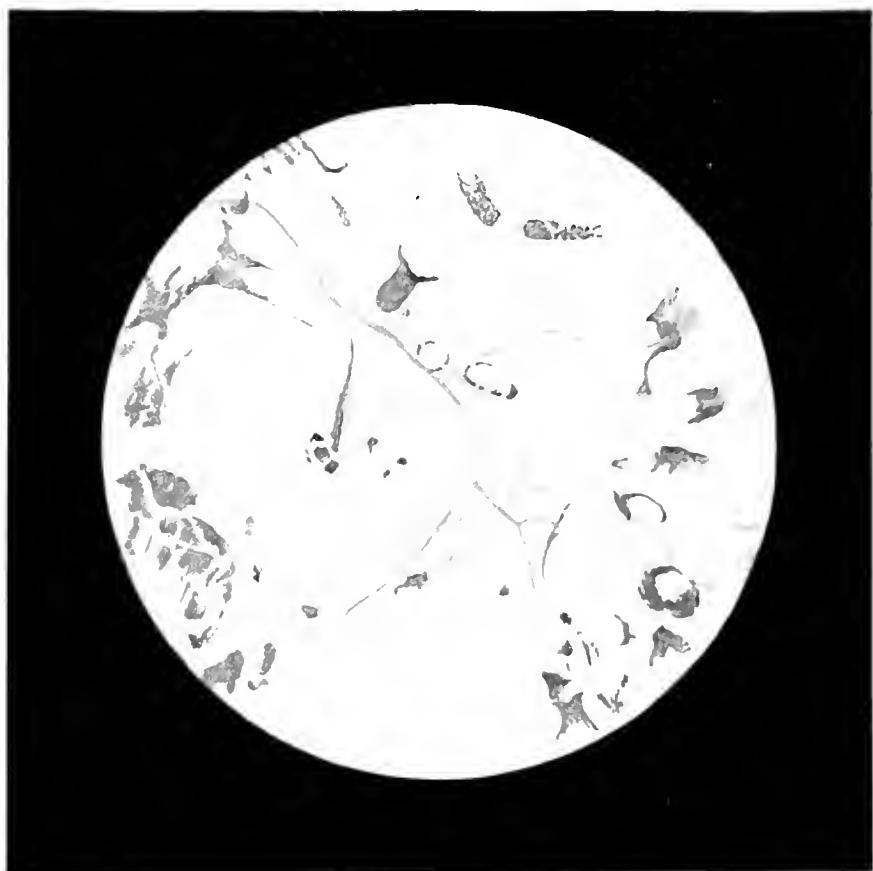


Fig. 76.



Fig. 77.

PLATE XLIII

Fig. 78.   Chorioretinitis Tuberculosa

## Fig. 78.  Chorioretinitis Tuberculosa

(See page 181)

This picture shows the typical condition of a tuberculosis of the chorioid that is fresh in some places, old in others.

The fresh tubercles cannot be seen, they make themselves manifest by the effect they produce on the retina; for the little, circumscribed spots are islands of retinal œdema.  It can be perceived that these are raised wherever a vessel of the retina passes over one; the vessel exhibits a wavy course at that place, as well as the loss of the light streak in the depressions, characteristic of changes of level.  The œdematous places are also a little hazy.

Part of the older places are marked by depigmentations, part by accumulations of pigment.  The wreathlike appearance is produced by the confluence of various individual foci.

An important point to be noted is that no vascular changes are to be seen in the picture.

Tab. 13.



Fig. 78.

PLATE XLIV

Fig. 79. Chorioretinitis Tuberculosa

# Fig. 79. Chorioretinitis Tuberculosa

This picture is one of an old tuberculosis. The bright spots are, almost without exception, produced by the confluence of individual tubercles. In the vicinity of these spots is to be seen a depigmentation that looks "as though a chemical fluid had been poured over them." The heaping of pigment in the vicinity is considerably less than in diseases of the chorioid that accompany sclerosis of the vessels. Pigment gradually migrates into the retina as it becomes atrophic (compare with Plate XXX).

The vessels of the retina and the papilla are normal.

Tab. 44.



Fig. 79.

PLATE XLV

Fig. 80.  Healed Inflammatory (Tuberculous?) Spot in the
Macula

Fig. 81.  Fundus of the Eye in Acute Miliary Tuberculosis

## Fig. 80.  Healed Inflammatory (Tuberculous?) Spot in the Macula

(See page 181)

An inflammatory spot about twice as large as the papilla had formed in the macula of a girl 14 years old.  Its etiology was obscure, but there were no signs of acquired or hereditary syphilis.  *Wassermann's* test proved negative, the tuberculin reaction on the contrary was positive.  The œdema of the retina disappeared under treatment with tuberculin, and the condition was left which is shown in the picture.  A white spot smaller than the papilla is to be seen, surrounded by a dark ring, and then by a lighter one in which the pigmentation is less.  In the neighborhood of the spot are three heaps of pigment of various shapes, but on the whole roundish, each surrounded by a brighter ring.

## Fig. 81.  Fundus of the Eye in Acute Miliary Tuberculosis

(See pages 129 and 132)

The margins of the papilla are very hazy, the veins are dilated, signs of an optic neuritis.  Three light gray spots are to be seen in the uniformly colored fundus: one round and with sharply defined edges, the others elongated and with indistinct margins.  The elongated spots have roundish, brighter nuclei, with a faded wing on each side.  The bright spots are tubercles in the chorioid, or, rather, patches of œdema in the retina that the tubercles excite by their presence.

Tubercles are particularly apt to lie at the posterior pole of the eye, and are almost always associated with an optic neuritis, which is caused by a meningitis.

This picture is from the eye of a boy 7 years old, who died of tuberculosis two days after it was taken.

Tab. **45.**
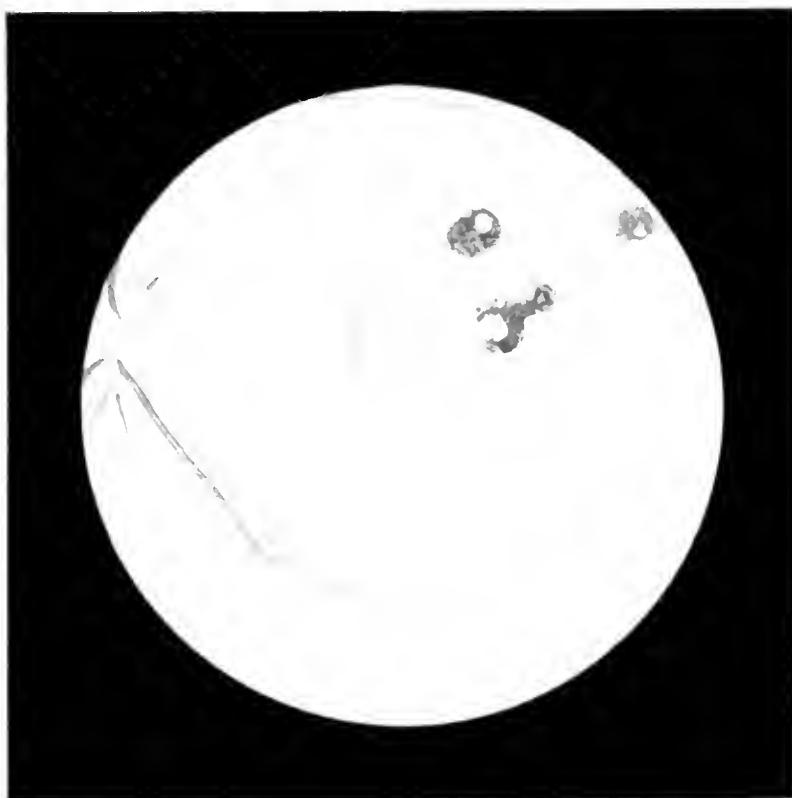


Fig. 80.
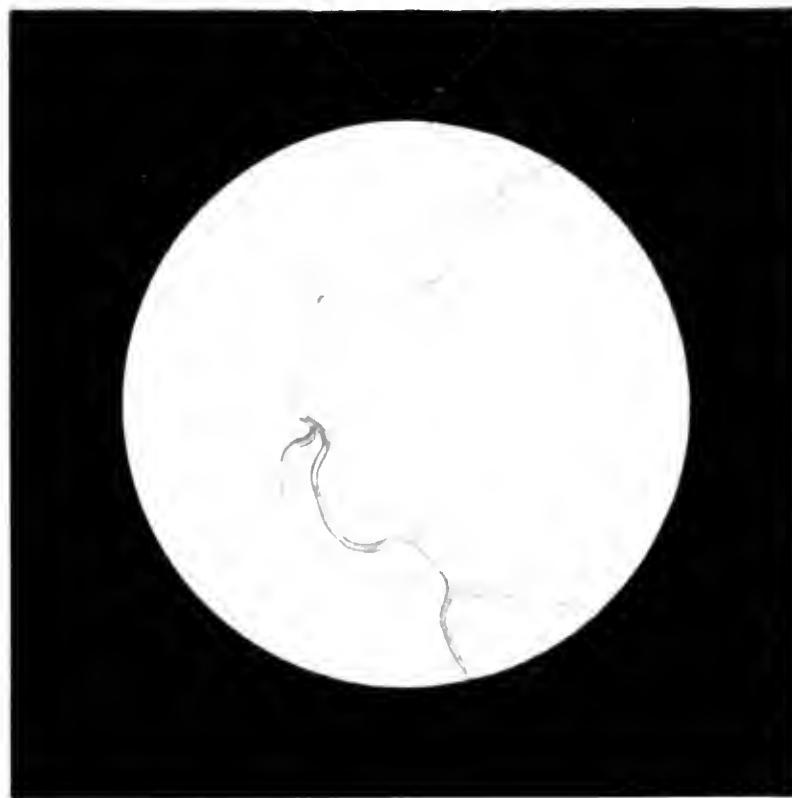


Fig. 81.

PLATE XLVI

Fig. 82.  Extensive Rupture of the Chorioid with Development
of Connective Tissue in Places

Fig. 83.  Rupture of the Chorioid

## Fig. 82. Extensive Rupture of the Chorioid with Development of Connective Tissue in Places

(See page 182)

This patient had received a blow on the eye from a broken belt. After the immense hemorrhage into the vitreous had been absorbed two large white spots could be seen at the place where the injury was received. One was a rupture of the chorioid with two points jutting to the right and the left. Traces of some vessels can be seen in the rupture. The entire place has an edge of pigment.

While the color of this spot is a light yellow, that of the other is rather bluish. Dots of pigment are abundant, both in the spot itself and in its vicinity. A distinct parallactic movement could be produced by moving the lens held in front of the ophthalmoscope. Close to this spot a smaller one is to be seen.

Both of these spots are to be regarded as *pure* ruptures of the chorioid, while the one between them shows distinct signs of a proliferation of connective tissue, which must be considered as a process of healing.

These ruptures of the chorioid at the place of impact are seen more rarely than the indirect rupture shown in the next picture.

## Fig. 83. Rupture of the Chorioid

(See pages 126 and 178)

A white crescent can be seen $2\frac{1}{2}$ P. D. from the papilla, to the margin of which it is parallel. The retinal vessels pass smoothly over its upper part, but make a little bend as they pass over its lower portion, perhaps on account of a commencing development of connective tissue. In the middle of the crescent is a little reddish spot, which may be either a hemorrhage, or a tuft of vessels. Its margins are distinctly pigmented.

Such ruptures of the chorioid, which often leave the retina intact, as it is more elastic, are the consequences of severe blows on the anterior part of the eye. In this case the cause was a blow with a billiard cue.

The effect on the vision varies according to the degree of injury to the retina and the position of the rupture. If the latter lies between the papilla and the macula, if the retina is torn, or if the rupture extends directly through the macula, vision will be badly impaired. In other cases it is hardly affected.

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Tab. **46.**



Fig. 82.



Fig. 83.

## Fig. 84.  Coloboma of the Chorioid

(See page 178)

Above the papilla in the inverted image, therefore in reality below, is a large, slate gray surface which has on one side a rather brownish tone. It has a broad edge of pigment. Within the surface, over which the vessels of the retina pass smoothly, are to be seen several bright stripes which are interpreted as folds. Some *scleral vessels*, twisted like corkscrews, enliven the picture farther in. The papilla and its surroundings are normal.

The vision depends on the extent to which the retina is involved; sometimes a defect is found in the visual field that corresponds to this place.

## Fig. 85.  Extensive Coloboma of the Chorioid

The coloboma, which in the preceding case stopped at some distance from the papilla, extends in this one above the entrance of the optic nerve, so that the disk lies in its area. The scleral vessels are very numerous.

In the middle of the coloboma is a roundish spot, the margins of which shift over its center when the observer moves the lens held in front of the eye. Hence there is at this place a depression, a so-called "sclerectasia." Some granules of pigment are scattered over the coloboma.

The rest of the fundus is of an albinotic character and so contrasts with those of the preceding cases.

Such colobomata are congenital and are not progressive.

Tab. **47.**



Fig. 84.



Fig. 85.

PLATE XLVIII

Fig. 86. Normal Fundus of a Rabbit

# Fig. 86.  Normal Fundus of a Rabbit

The papilla is transversely oval and is plainly excavated, or surrounded by a wall over which the vessels of the retina rise.  To the right and left of the papilla are two enormous white wings of medullated nerve fibers in which the vessels of the retina course.

The rest of the fundus is uniformly colored.

Tab. 48.



Fig. 80.

# Index

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